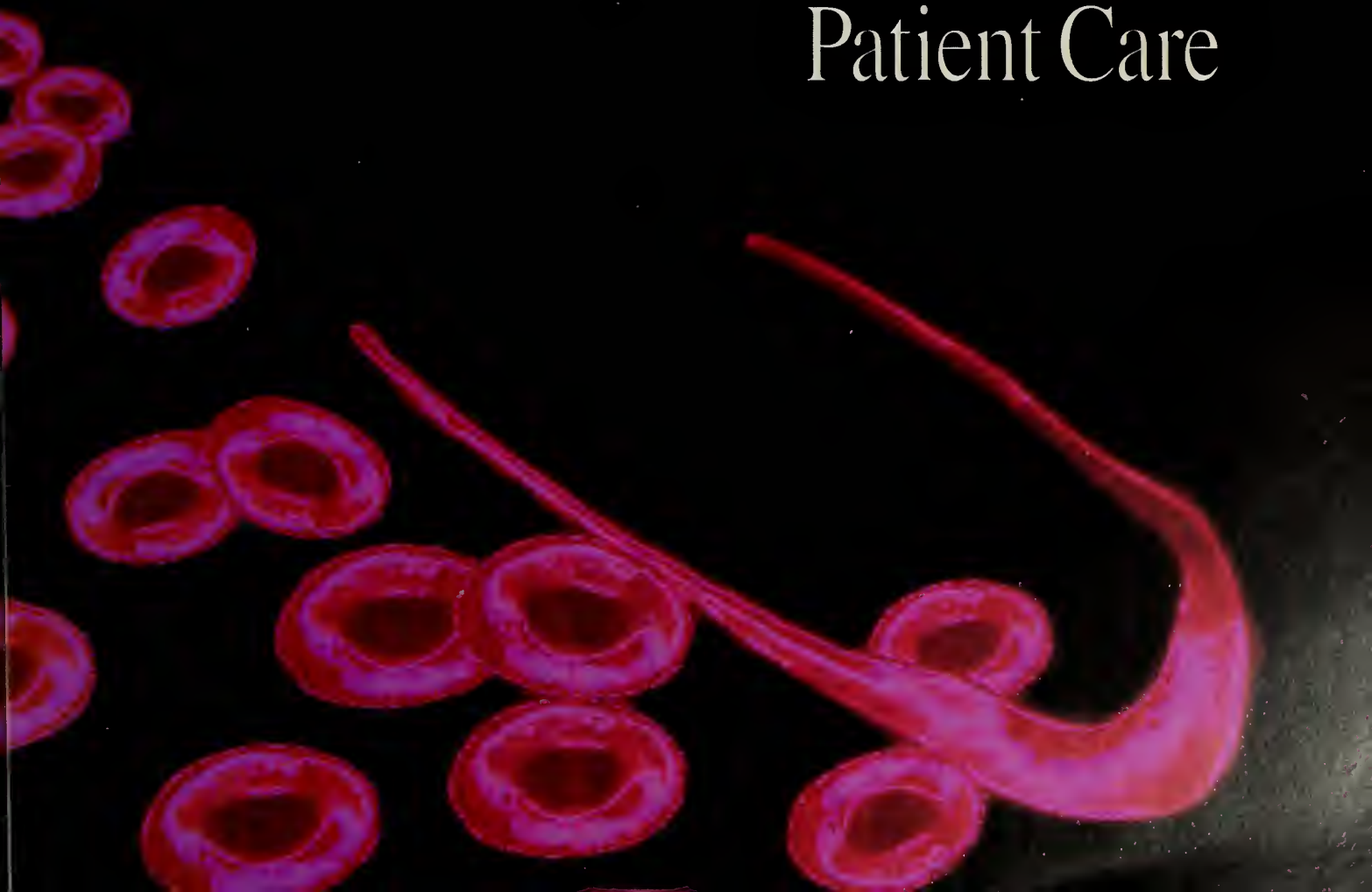
THE MEDICAL COLLEGE OF GEORGIA
The Health Sciences University of the State of Georgia

Today

USPS 867340 Volume 19, Number 2, Winter 1991

Sickle Cell

Research
Patient Care



Introduction

If you ran into Lindy Riles, Terry Key or Cocoa Winfrey on the street, you wouldn't notice their disease. They certainly look healthy; nothing about their appearance betrays the many maladies that can accompany sickle cell anemia.

But those maladies—which run the gamut from debilitating pain to cirrhosis of the liver to stroke—are all too real. And Lindy, Terry and Cocoa are all too familiar with them. So are the thousands of others in the region who have the incurable hemoglobin abnormality resulting in sickle-shaped cells that clog arteries and impede oxygen delivery.

But despite the myriad of health problems associated with the disease, sickle-cell patients are living longer and better than ever. And the Medical College of Georgia Comprehensive Sickle Cell Center has made world-renowned contributions to their care.

This edition of *Medical College of Georgia Today* introduces you to the center, its researchers and clinicians and the patients it serves, including Lindy, Terry and Cocoa.

Read about how the center is improving their lives. For instance, MCG has tested a drug that increases the body's level of fetal hemoglobin. The higher the level of fetal hemoglobin, the milder a case of sickle cell disease will be. Read also about a new method of relieving pain, a common companion of the disease. And learn how MCG researchers are helping prevent young sickle cell patients' strokes.

Also featured is information about one of the most virulent hemoglobin disorders related to sickle cell disease: thalassemia. MCG is an international reference center for identification of its many different forms.

And this edition wouldn't be complete without featuring the center's founder and director, Dr. Titus H.J. Huisman. He'll step down from the post soon, but his research will continue and his contributions to the school will live forever.

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The Medical College of Georgia is the health sciences university of the University System of Georgia. Focusing on health care education, research and patient care, the Augusta-based institution consists of MCG Hospital, more than 80 support clinics, statewide outreach programs and the Schools of Allied Health Sciences, Dentistry, Graduate Studies, Medicine and Nursing.

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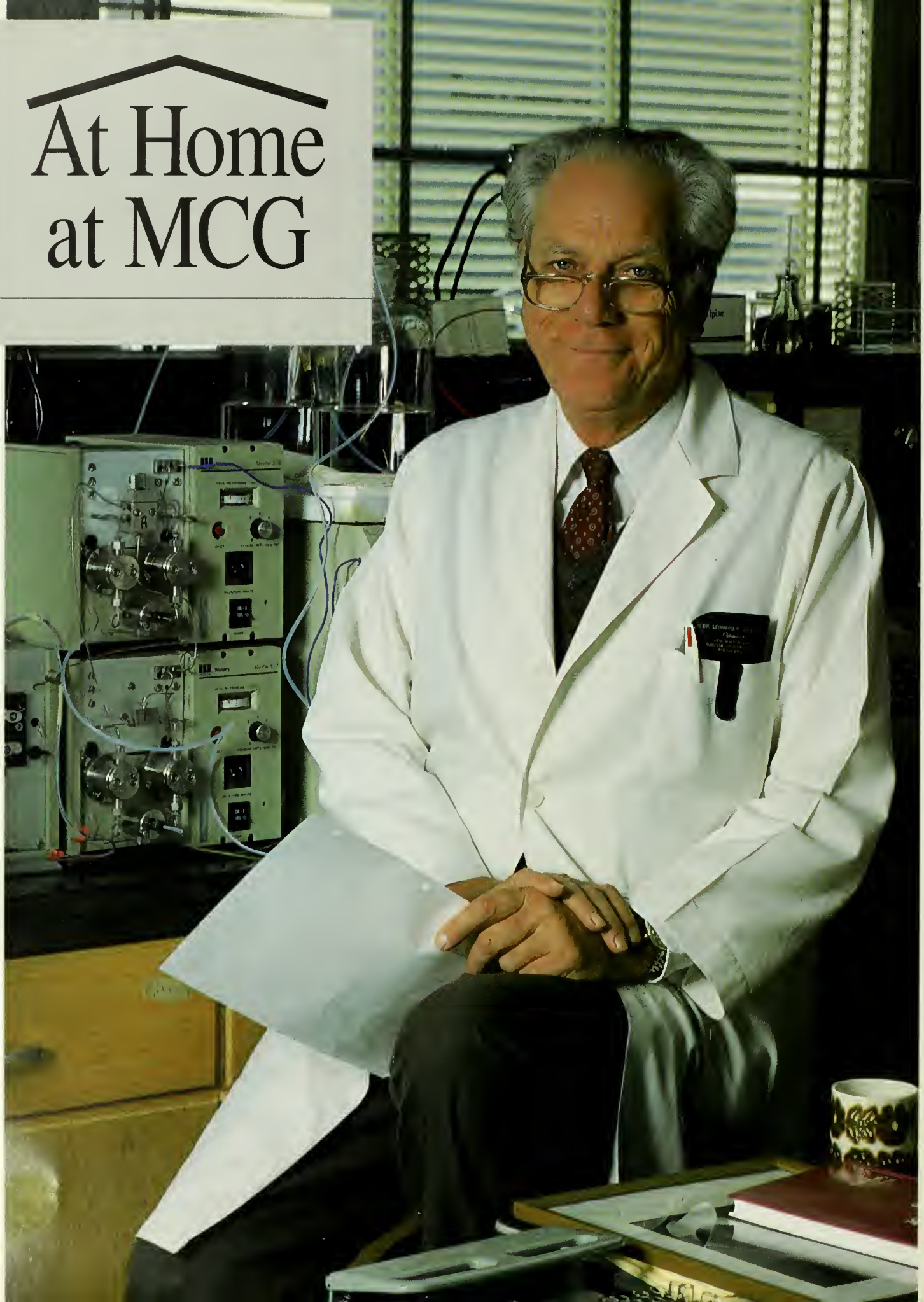
At Home at MCG



When a young scientist from the Netherlands packed up and moved with his wife and two daughters to Augusta in 1959, he anticipated only a brief stay—two years, tops.

He never would have guessed that he, who so carefully plots every step he takes, was stumbling onto a whole new life.

He never went back home. More than three decades later, that scientist is still in Augusta. He still considers the Netherlands home, and a thick Dutch accent betrays his roots. But despite his perception of himself as something of a perennial visitor, no one is more at home at the Medical College of Georgia than Dr. Titus H.J. Huisman, Regents' professor and chairman of the Department of Cell and Molecular Biology and director of MCG's Comprehensive Sickle Cell Center.

Dr. Huisman, whose work to a large extent is his life, should have known he would never walk away from a job when more work has yet to be done. And he's had his work cut out for him at MCG.

Dr. Huisman, 67, earned a Ph.D. in chemistry from the University of Groningen in the Netherlands and a doctorate in science from the University of Utrecht, also in the Netherlands. After graduation from the latter, he headed the biochemical research laboratory in the University of Groningen Department of Pediatrics from 1951 to 1959. While there, his interest intensified in hematological disorders.

"I saw my first sickle cell patient in Detroit in 1955 while on faculty at the University of Groningen," he said. His research in the pediatric department acquainted him with fetal hemoglobin, the blood product that largely diminishes and gives way to adult hemoglobin as people age. But the amount of fetal hemoglobin that one retains varies from person to person. This fact intrigued Dr. Huisman. What determines how much one retains, he wondered? The answer held more than academic significance. The more fetal hemoglobin a sickle cell patient retains, the milder his disease will be.

The Netherlands itself had few cases of sickle cell disease as Dr. Huisman began probing the disorder, although some of its citizens in colonies in the West Indies were affected. Therefore, Dr. Huisman was more than a little interested when he received an invitation from Dr. W. Knowlton Hall, then chairman of the Department of Biochemistry at MCG.

"Professor Hall asked me to join the faculty because he knew I was interested in hemoglobin," Dr. Huisman said. He accepted the invitation; in 1959, Dr. Huisman and his family moved to Augusta, where he planned to set up a lab at MCG and leave two years later.

"It was a tremendous change, coming from an old university in the Netherlands to this medical school," Dr. Huisman said, noting considerable culture shock upon arrival. "Strange, strange, strange people..." he said of his initial impression of Southerners. "Very strange."

But work was his main preoccupation. He found himself smack in the middle of an area rife with sickle cell disease among the black population. Georgia and South Carolina have about 5,000 cases of sickle cell disease, and 100 children are born with it every year in Georgia. "All the cases are severe here," he said. "I can say that with almost no exception."

That fact added a certain urgency to his work, and Dr. Huisman wasted no time. In 1960, he was awarded a National Heart, Lung and Blood Institute grant to establish a hematology laboratory at MCG.

"And I still have that grant," he said proudly. "That must be a record. My 31st year."

In 1972, a National Institutes of Health grant for the study of abnormal hemoglobins enabled MCG's establishment of a Comprehensive Sickle Cell Center, "one of the few in the country," said Dr. Huisman, who became the center's director.

Since then, the center has incorporated the work of faculty from various departments—medicine, pediatrics, cell and molecular biology and neurology, to name a few. The center also has attracted fellows from throughout the world and is a reference center for blood typing and testing with an international reputation.

And although a cure still eludes the medical community, Dr. Huisman and his colleagues have spearheaded research and patient-care techniques which have added immeasurably to the quality of life of those with sickle cell anemia and related disorders.

"We know the gene; we know what the mutation is in the gene that causes sickle cell. We know now that sickle cell has originated independently in at least five different places," Dr. Huisman said, noting that varying chromosomal mutations differentiate one form of the disease from another.

He has traveled the world to gain more insight into the disease and its variations. "In India, the disease is very mild because fetal hemoglobin is so high," he said. "In Nigeria, the cases are much worse because the fetal hemoglobin is very low. We (researchers) are still arguing among ourselves if there is a different mutation in the Mediterranean."

A common myth about the disease, he said, is that it targets only blacks. "It's a very big misconception that sickle cell is a black disease. One out of 10 people in the world have a hemoglobin abnormality. It just happens to occur more with blacks than with whites." Dr. Huisman thinks the reason is that blacks have traditionally populated regions most heavily hit by malaria. Sickle cells are particularly resistant to malaria, so the hemoglobin disorder has actually been a protective device in those cases.

"Where malaria is rampant, sometimes more than 25 percent of the population has sickle cell," Dr. Huisman said. "It's a tremendous advantage to have a sickle cell gene if you have malaria."

New ground is being broken continually about the disease. For instance, "It's now possible to introduce the sickle cell gene into an animal model," Dr. Huisman said. Also, the disease now can be diagnosed prenatally. And bone marrow transplants have been used with some success to treat thalassemia, a particularly virulent hemoglobin disorder.

And even though science hasn't completely caught up with sickle cell anemia, those with the disease can live full lives, Dr. Huisman stressed. "The quality of life is quite good for some. The key is good medical care. They can live to their 70s."

Dr. Huisman retires in January as chairman of cell and molecular biology, a post he held for 14 years, and he is searching for a successor as director of the center. He plans on continuing his work as a professor in the newly created Department of Biochemistry and Molecular Biology and as editor-in-chief of *Hemoglobin*, the international journal of hemoglobin research. After that, his plans aren't entirely finalized. "I don't know what I'll do when I retire. I might become an artist; I love to paint. My wife might think it's terrible to have me home all day, so I might continue to work as an emeritus."

Or he might move back to the Netherlands, the home he never intended to leave. But Augusta is home now, too. "You form a group of people and a group of friends... That's very gratifying," he said. "I've always enormously enjoyed my work. I've no regrets. And I must have liked Augusta, because I've been here for 31 years. I'd planned on staying for two."

—Christine Hurley Deriso

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Once she had it, she had a 75 percent chance that the disease might not be too awful.

But at 31, with six major operations, including one to halt destruction of her young hip, another gallbladder surgery that actually required three procedures before it was over, more than 60 hospitalizations for the severe pain crises she likens to a simultaneous throbbing ache in every tooth in her head, she obviously beat the odds—for the worse—once again.

"When I was younger, I didn't like to tell people. When I did, they used to say I was going to die." Her older brother, John, did die from the disease when he was 30 and she was still a teen-ager. Her living older brother, Michael, also has the disease.

So when Ms. Riles turned 18, she finally asked her mother if she was going to die. Her mother said no, not until her time. "I just started telling people I was going to live, just like they are."

She does live.

But it's with a debilitating pain that can flare at any time and last for weeks at a time, a pain so familiar that she always knows when it's going to be bad enough to head for the hospital; a pain that has kept her from being able to complete college so she can be the teacher she wants to be; a pain that has no promise of an end.

But she also lives with her bright, 7-year-old daughter Nadia who gives her alcohol rubs when she knows she feels bad. Nadia has sickle cell trait. As Nadia grows older, Ms. Riles will talk candidly with her daughter so that she completely understands the risks of bringing a child with sickle cell disease into the world.

Even though sickness forced her to discontinue her own studies—at least for now—Ms. Riles spends some of her days at Nadia's elementary school working with the children. She also loves to care for her friends' children while the friends are at work.

"You learn to accept it. That's what you are, you can't do anything about it," Ms. Riles said. "I've got a beautiful little girl. I've got a wonderful family. So, I'm doing fine. I'm just glad I'm here."

Dr. Paul Milner sees Ms. Riles at least twice a month as he tries to keep two steps ahead of the disease he's been battling all his professional life.

Back in 1968, he was up to his elbows in sickle cell disease as chief of hematology at the University of the West Indies in Jamaica. He started a clinic there for the thousands of patients that no one was looking after.

His interest took him to Johns Hopkins University to study a new treatment using cyanate, a poison that would alter patients' hemoglobin and prevent sickling. But the side effects were awful. The treatment was abandoned. He went back to Jamaica.

Time and his interest in sickle cell disease soon brought him to the Medical College of Georgia.

1978 found him enrolling Ms. Riles and 159 other patients at MCG in a decade-long national study designed to get good, solid data on this troubling disease.

"When we went into this study, people were talking about the life expectancy was 20 years, that there wasn't much point in doing anything for them because nobody lived," said Dr. Milner, professor of medicine and pathology and physician in charge of the Adult Sickle Cell Clinic at MCG.

He knew that philosophy was wrong.

And the 10-year National Institutes of Health study of 4,000 patients was to provide the hard data to back him up.

MCG's 160 study participants were ages 15 to 60. "We looked at everything that happened to them," Dr. Milner said. "We looked at every admission and what it was for. We X-rayed all of their hips and all of their shoulders looking for bone problems. And then we X-rayed them again five years later, the ones that didn't have problems and we found a lot of new problems. So we got into the incidence of complications."

The study documented the uncomfort-

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able minority Ms. Riles finds herself in, that 25 percent of all patients with sickle cell disease who get frequent pain crises.

"People who know about sickle cell disease realize that the patients that they see in the hospital are the same ones over and over and over," he said.

It's odd, this genetic disease that turns donut-shaped red blood cells into banana-shaped cells that no longer do an efficient job of carrying oxygen to the body but instead clog tiny arteries and impede oxygen delivery.

Varying forms and severities of this disease exist.

One form means a patient's own body is so efficient at destroying the sickled cells that patients rarely have pain crises, in which vessels are clogged and oxygen-starved tissues respond with pain. But these patients live with chronic anemia and fatigue.

Another form makes the sickled cells more durable, so there's relatively little anemia, but they have more pain crises and damage to hip bones and shoulder bones.

Another form seems to focus its destruction on the delicate vasculature of the eye. "I have patients who are blind in their 20s from being neglected," Dr. Milner said.

"The study found that sickle cell anemia is a very diverse condition. We know an awful lot more about what to expect from sickle cell disease than we knew before. It's not as bad as it was made out to be in the literature for a lot of people. Some people seem to live quite happily with it and do quite well and don't go to the hospital very often. Others are severely debilitated from early childhood."

But among the 25 percent most affected by the disease, there are deaths and some are really unexplained. "Patients come into the hospitals and they died and at autopsy nobody would say why they died," he said.

"So we know that these people are at high risk when they come into the hospital because of biochemical disturbances and susceptibility to infection. So they need very careful care. They can be walking around perfectly well one minute and really seriously ill the next day and people may not appreciate that. We know that happens because their chronic anemia can turn into a severe condition by slight changes in their biochemical environment, like severe dehydration and acidosis."

Others die more predictable deaths, like cirrhosis of the liver. The liver is a very vascular organ whose vessels clog, causing the organ to get bigger and bigger until it finally fails. The liver also can be destroyed by hepatitis resulting from multiple transfusion. Dr. Milner lost two patients to liver failure during the NIH study.

Hearts suffer, too, as the blood vessels of the lungs gradually become blocked and the right side of the heart gets bigger and bigger pumping against this resistance until the heart fails.

Patients can end up on dialysis or even die from the complications of the sickled cells clogging up the vasculature of the kidneys.

The hard data gained from the first decade of study prompted centers like MCG to participate in a second, five-year study now under way following patients 35 and older who were in the original study.

This study will focus on the now-documented causes of death, the kidney, liver and heart failures, and follow these older patients to chronicle complications, and for some, their deaths.

"There are a lot of things that can come out of studying a group in a prospective way," Dr. Milner said. "On the whole, what it amounts to is that these people need to be in a program where they can get regular physicals at six-month intervals so that problems can be seen ahead of time and prevented."

—Toni Baker

Stacking Kids' Odds

It's that time of month again.

Terry Key knows the routine all too well, but he still cries when his mom tells him it's time for his appointment.

"Don't cry, baby," Virginia Pollard says soothingly to her 8-year-old son. "This will help you. You'll feel better afterward."

So Terry dries his eyes and climbs into the car. He and his mother head from their Aiken, S.C., home to the Medical College of Georgia outpatient building. Once there, he enters a cheerful, brightly decorated room and is greeted warmly by the staff. Then he offers his tiny arm to a nurse and winces as a needle pierces his skin. He'll sit there for the next two or three hours. It's time for his monthly blood transfusion.

Terry, a quiet child with an endearing smile who would much rather discuss Teenage Mutant Ninja Turtles than his health, has known pain all his short life. He has sickle cell anemia, a genetic disease characterized by an abnormal red blood cell with a defective form of hemoglobin that causes the cell to become sickle-shaped when deprived of oxygen. Because of the disease, Terry spent his first four years of life plagued with debilitating pain crises. He and his mom had, sadly, become used to them.

When Terry was 4, the condition took an even greater toll. Terry was playing quietly at home when he suddenly experienced what Ms. Pollard assumed was another pain crisis. He cried in anguish and passed out briefly. Once the incident had passed, Ms. Pollard knew it hadn't been an ordinary pain crisis. Her son's right side was paralyzed.

She took Terry to his doctor, who told Ms. Pollard to hospitalize him immediately. Hospital tests confirmed the doctor's suspicion: Terry had had a stroke.

Ms. Pollard was devastated, and even more so when the bad news compounded: it wasn't the first stroke Terry had experienced, doctors told her. The tests indicated he'd had several previously, but they were mild enough to go unnoticed.

"I felt like a part of me died when I heard about those strokes," she recalled. "I thought, 'God, I'm right here with this child. I should know what's going on with him.' It made me feel like I did something wrong."

But Ms. Pollard isn't at fault. Much about strokes experienced by children with sickle cell anemia stymies even the medical community.

"About 5 to 10 percent of sickle cell patients are at risk of stroke," said Dr. Robert J. Adams, associate professor of neurology at the Medical College of Georgia.

But which 5 or 10 percent? And what exactly causes the strokes? Dr. Adams is beginning to find out. "Our research grew out of discussions with (MCG pediatrician) Dr. Virgil McKie and developed out of the frustration of him and his colleagues in pediatric hematology of not being able to either prevent strokes or know who should get preventive treatment." That treatment is a regular blood transfusion.

Dr. Adams' research, funded by a five-year, National Institutes of Health grant encompassing several sickle-cell projects at MCG, seeks to answer those questions. He is trying to develop a non-invasive method to determine which sickle cell patients are at risk of stroke and to find out more about the nature of the strokes themselves. Dr. Virgil P. Sydenstricker, a now-deceased former chairman of the Department of Medicine at MCG, was the first to report the stroke risk of sickle cell patients. Since that time, some half a century ago, much as been learned about the subject.

Sickle cell patients have oxygen-deficient blood. Therefore, their bodies rush blood quickly to the brain to compensate for the deficiency so the brain will have adequate oxygen. The blood flow to the brain is about 50 percent faster in sickle cell patients than in that of non-anemic people.

Children at risk of stroke have scars on the arteries supplying the brain. Doctors speculate that the scars result from injuries caused by turbulence produced from overly fast blood flow rates or from the contact of the sickled cells with the vessel wall. "We think the arteries become narrowed due to a scarring process that progresses over months to years," Dr. Adams said.

Such narrowing of the vessels increases flow rate still more. Dr. Adams offered an analogy: "If you're on an interstate with five lanes that goes down to two lanes, you've got to really increase the speed in those two lanes to keep the traffic flowing smoothly," he said. But what if the traffic doesn't maintain its flow? What if traffic—or in this case, the blood flow to the brain—jams? Infarction, blockage of blood flow to the brain, can result, and a stroke can follow.

The process is called endothelial hyperplasia, but much about the condition is unknown. "In essence, we do not know why these scars occur or why one child with sickle cell has narrowed vessels and another does not."

Dr. Adams and co-investigators from adult and pediatric neurology, radiology, pediatrics and cell and molecular biology are trying to find out by concentrating both on the increased blood flow and an unknown factor that he suspects also contributes to stroke risk. "We think there's something about the red blood cells themselves or something in the blood serum," he said. "It takes the severe anemia plus this unknown factor to create the stroke risk." He is trying to develop an animal model to help determine that unknown factor. "We ultimately want to know why the lesions develop," he said. "That's the key." If they find it, researchers will much better understand the stroke process, the first step in developing treatments that will put them on the offense rather than the defense.

Terry Key undergoes transfusion.

The present treatment—regular blood transfusions—helps enormously and is a potential lifesaver, but it has drawbacks. For one thing, blood transfusions are uncomfortable. "Terry hates being stuck all the time," his mother, Ms. Pollard, said. They're also time-consuming. Ms. Pollard has to plan extensively around their monthly treks to Augusta. "You have to make a day of it," she said.

Of course, it's worth it: transfusions decrease stroke risk by lessening vessel narrowing and also reduce pain crises. "After a transfusion, Terry has more energy than a normal child," his mother said.

But regular transfusions carry their own risks, including iron overload. And doctors aren't sure when to eliminate the treatment. Children tend to outgrow the risk of infarction at about age 15. "But it's not clear when to stop transfusion," Dr. Adams said. "It's a serious problem. There isn't a good guideline to go on."

He hopes the medical community will eventually understand sickle-cell strokes well enough to offer alternatives. "And finding the unknown factor also might shed light on strokes in general," Dr. Adams said.

In the meantime, they are concentrating on stroke prevention. "What doctors have been forced to do is wait for children to come in with stroke symptoms, which is unsatisfactory," he said. "The damage is already done." Stroke damage can produce learning disabilities, behavioral problems, spasticity or incoordination. It also results in paralysis, as in Terry's case, although physical therapy has helped him regain much of his strength. "And we know that unrecognized minor strokes occur that may take their toll cumulatively," Dr. Adams said.

To prevent the damage caused by these strokes, the research effort is to identify children at highest risk so treatment can begin before the first stroke.

The key to his success is an instrument, developed within the past five years, called transcranial Doppler, or TCD. A TCD is a non-invasive, portable ultrasound device that measures blood flow speed in arteries. It is painless, risk-free and relatively inexpensive.

Other technology is also available to ascertain brain damage or vessel narrowing, including magnetic resonance imaging (MRI) and angiography. But both are too expensive to be practically used as screening devices, and the latter is invasive.

Dr. Adams and his co-workers refined TCD's usefulness by testing blood flow speeds of healthy children. They then tested speeds of children with sickle cell anemia and determined rates that are normal for anemic children, even though the speeds are faster than usual. "If we test a child and his blood flow is even faster than that, then we assume the child has injury sites that make him at risk of stroke," Dr. Adams said.

Because of TCD's many advantages, his goal is to use it to test the blood flow speed of every child with sickle cell anemia. "Then, if the test indicates stroke risk, magnetic resonance imaging can verify the injury sites," he said. "Our goal is three-fold: to develop TCD as an effective screen; to develop and improve MRI to follow up TCD in abnormal cases; and to determine whether unnoticed strokes have already occurred. This makes it feasible to contemplate primary stroke prevention. That was never feasible before."

All MCG sickle-cell patients age 5 to 16 are now tested, including those in outlying clinics. "We do about 10 to 15 tests a day," said Betsy Carl, research coordinator in the Department of Neurology. "The test lasts about half an hour. Then, we bring the kids with (high blood flow speeds) back to MCG for MRI."

Dr. Ramon Figueroa Ortiz, assistant professor of radiology, is spearheading the MRI contribution to the research project. MCG reported the first experience with brain MRI several years ago. As new equipment and software techniques are developed, it may be possible to verify the arterial scars without angiography and follow their condition using MRI.

If injury sites are ascertained after the children are tested, blood transfusions begin immediately.

Dr. Adams wants to duplicate the procedure for every anemic child. "The goal is to screen all sickle cell children," he said. "We're trying to blaze the trail for other sickle cell clinics."

—Christine Hurley Deriso

Easing the Pain

A new approach to dealing with the agonizing pain crises experienced by people with sickle cell anemia is netting better pain relief and shorter hospitalizations using less narcotic drugs, according to doctors at the Medical College of Georgia.

MCG doctors are now treating these pain crises with patient-controlled analgesia pumps.

With these pumps, patients give themselves periodic doses of pain medication designed to even out the peaks and valleys of pain relief offered by the more standard approaches to managing pain.

"It's a terrific improvement," said Dr. Paul Milner, professor of medicine and pathology and physician in charge of the Adult Sickle Cell Clinic at MCG.

He describes the pain some sickle cell patients experience "like being kicked on the shin by a horse and that pain going on and on and on."

Crises occur because this genetic disease results in abnormal hemoglobin, the oxygen-carrying component of the blood, Dr. Milner explained.

Hemoglobin travels through the body in the red cells and these cells are made stiff by the abnormal product they carry, the sickle cell hemoglobin.

Problems occur in the tiny vasculature of the bone marrow where blood is produced.

Numerous, tiny capillaries are actually smaller than the red cells, so the red cells must deform to pass through them. But the stiff red cells can't deform properly and sometimes get stuck in the tiny blood vessels.

Pain results from lack of adequate blood and oxygen to the bone.

Ironically some sickle cell patients do not get these crises because they are so anemic, Dr. Milner said. But the extreme anemia of these patients can result in a general feeling of fatigue; persistent ulcers on their shins; and, as they get older, progressive kidney and heart failure.

But about 25 percent of all patients with sickle cell anemia experience debilitating pain attacks and the patients and doctors who care for them quickly learn who falls into the 25 percent.

"That means in any given hospital you see the same patients over and over again," Dr. Milner said.

"When we first see the patient, they are usually agonizing, curled up and crying," said Dr. Rudolph H. de Jong, professor of anesthesiology and director of the MCG Pain Management Center.

Standard therapy for these patients includes pain medications given by shot or intravenous drip for the three to six days these patients average in the hospital with each major pain crisis.

This approach results in a rise and fall of drug levels in the body with two-thirds of the time the patient getting too little or too much drug, Dr. de Jong said.

"What we are trying to do with the patient-controlled analgesia pumps is to get this patient at a steady drug level, neither too little nor too much," he said.

Doctors began using the pumps at MCG Hospital and Clinics primarily to help manage pain following surgery and Caesarean sections.

Dr. Milner approached the pain management team because he reasoned that the same approach might be useful for his patients who were the most difficult to manage in crisis.

Initial assessments by pain management and sickle cell disease doctors show that the patients are getting better pain relief and requiring shorter hospital stays, both with less medication.

When patients first come to the hospital in crisis, they are immediately given a shot with enough pain killer to ease the crisis. Then doctors begin to calculate just how much drug will be needed to keep the patient comfortable.

The pumps are set to deliver a baseline dose of drug and then patients can give themselves a small booster dose when they begin to feel discomfort. Pumps are pre-set so a patient can't overdose.

"From talking to Dr. Milner, we get the feeling that these patients do recover faster," Dr. de Jong said. "We get them back to school, back to normal activity quicker than we could in the past. Patients certainly like it and feel comfortable with it. I think it's going to catch on," he said of the pioneering approach to managing sickle cell disease pain crisis.

—Toni Baker

Instant Access to MCG

The expertise and assistance of Medical College of Georgia faculty is only a phone call away, thanks to the MCG Physician Consultation and Referral Service.

The service, which began in December, is in response to many requests from alumni and other referring physicians. It offers a toll-free number to help practicing physicians reach MCG faculty physicians. Physicians who call the MCG Consult Line will speak with an MCG representative who will quickly connect them with the appropriate faculty member or service. The phone number, accessible seven days a week, 24 hours a day, is 1–800–733–1828.

A call to the consult line will enable a physician to, among other things:

—receive consultation on a difficult-to-manage case;
—discuss diagnosis and therapy;
—ask about new patient care methods;
—explore the latest research findings;
—discuss a patient referral;
—follow up on patients hospitalized or treated at MCG;
—obtain information about MCG continuing medical education activities.

"This service will facilitate access to our faculty for any physicians who wish a consultation," said hospital director R. Edward Howell. "The representative answering the phone serves as a resource person to match the calling physicians with the appropriate source of information at MCG."

"The callers may need access to our expertise or just to talk things over," said Dr. Gregory L. Eastwood, dean of the School of Medicine. Many rural hospitals, for instance, lack specialists in various areas. A physician confronted with an unusual case may request consultation with an MCG representative. Or he may want to refer the patient to MCG. This program will facilitate the referral process.

Dr. Eastwood said he hopes the program will be a major step toward enhancing physician communication. "It's very important to develop programs to improve communications among physicians throughout the state and our faculty," he said. "And one of our major missions is to make MCG known in the Southeast as a place of excellence for patient care. This program will indicate to physicians what we have to offer."

Another aspect of the service is a directory of MCG clinical faculty and administrators which will be available soon. "The *Directory for Referring Physicians* will supply information regarding faculty members, services and departments," said Diane Barnard, manager of the service.

MCG is confident the program will provide a much-needed service for the physicians of Georgia and the region.

AlumNews

Ribbon Cut on Old MCG Renovation Project

Christine Hurley Deriso

Some 200 people poured into the Old Medical College Nov. 30 to celebrate the building's renovation and reopening.

The building gleamed like new with freshly painted white walls and shiny hard-wood floors, but its history was the order of the day for the 10 a.m. ribbon-cutting and open-house ceremony.

"Whether you're a new Augustan or have lived here a long, long time, this building represents the history of our community," Augusta Mayor Charles DeVaney said during his proclamation that formally recognizes Nov. 30 as Old Medical College of Georgia Day. "Once a building is gone, we can never replace it. Isn't it wonderful to have this right here with us in usable condition."

The intention of the approximately $2 million renovation project was to restore the 155-year-old building, MCG's first home, as closely as possible to its original appearance. The MCG Foundation, which leases the building from the Academy of Richmond County, spearheaded the fund-raising and organizational efforts necessary to complete the project.

Dr. Francis J. Tedesco, Dr. H. Gordon Davis and Jack Hagler cut ribbon on "new" Old MCG.

Dr. Francis J. Tedesco, president of MCG, lauded the efforts of one foundation member in particular during his remarks at the ceremony. "There were a lot of times when progress stopped (on this project)," Dr. Tedesco said. "Only one person allowed the project to continue: (John C.) Jack Hagler." He presented Mr. Hagler, chairman of the foundation's Old MCG restoration committee, with an MCG watch.

Dr. H. Gordon Davis, president of the MCG Foundation, noted in the ceremony's opening remarks that 2,345 MCG students graduated in the building. "From this building, the Medical College of Georgia has grown into a great university," he said. "The university will always be symbolized by this building." He thanked the Academy of Richmond County for letting the foundation restore the building.

Dr. Harold Nelson, president of the Richmond County Medical Society, also spoke during the ceremony, noting that the society's affiliation with and support of MCG date back to the early 1800s.

The restored building will be used as a continuing-education center and for such MCG functions as homecoming and reunion dinners, alumni gatherings and official ceremonies with students and faculty. The building also will be reserved for community and statewide functions, including cultural events, social affairs, dances, banquets and weddings. Still to be completed are construction of an elevator and restoration of the building's exterior and gardens. ■

Anthropologist Digs Into Old MCG's Past

Christine Hurley Deriso

A black wrought-iron gate creaks wearily as it opens onto the backyard grounds of the Old Medical College building. On those grounds, the limbs of towering oaks and elms stir lazily in a hot summer breeze. An imposing brick wall, chipped with age yet as strong as the day it was built, fences off the property and shields it from the bustling city just beyond.

That back yard seems a world unto itself, a small piece of history stubbornly clutching its roots.

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Friends of MCG gather to celebrate reopening of Old Medical College

Old MCG... *continued from page 11*

Indeed, those roots extend deep into the past, yet are very much alive. And a Georgia State University anthropologist wants to learn as much about those roots as possible.

Dr. Robert L. Blakely, associate professor of anthropology at Georgia State, spent the first two weeks in September in Augusta, along with nine of his students, to literally dig into the back yard's past.

It wasn't his first trip to the Old MCG building. He was on the scene last year after the building's renovation uncovered long-buried human bones. Ever since, he's considered the site to be an anthropological goldmine.

On this trip, he confined himself to the building's back yard—the site of three long-gone buildings. "What we essentially came looking for were building foundations constructed on the grounds in the 19th century," he said. "We have crude maps from the late 1800s that show buildings on the property that are no longer here. We wanted to locate exactly where they were."

One building was the city morgue, known as the "dead house." "We think we're beginning to find evidence of it," Dr. Blakely said.

The other two buildings—a dissecting room annex and City Hospital—are literally right under foot. The top several inches of ground in the back yard is made up of rubble left behind when the buildings were demolished in 1930. "That rubble has built up the ground," Dr. Blakely said, noting that some of the buildings' bricks were recycled. The wall that fences in the grounds, for instance, was constructed from the buildings' bricks.

He had only to dig about a foot deeper to discover the foundation for the annex, which was built in 1837 as a dissection laboratory for MCG students. "Very soon after the Old MCG was built, they realized they needed a lab for dissecting, so they built a long, narrow building called the dissecting room annex," Dr. Blakely said.

In 1869, City Hospital was built, which adjoined the annex in a "T" shape.

The remnants of the buildings are providing insight into aspects of the 19th century. "Part of our goal is determining styles of architecture," he said. "Although we know these buildings existed, we know very little about what they were like." But Dr. Blakely is finding out. "One thing that surprises me is how solid these structures were. They were built to stay."

He and his students also are uncovering other clues about the period. They've dug up various artifacts, including medicine bottles, buttons and animal bones. All the material will be taken to Georgia State for investigation, then will be returned to the MCG Foundation.

"We've recommended that the foundation build an exhibit in the Old MCG to tell the story we've uncovered," he said.

As Dr. Blakely wrapped up his mission, during which he and his students slept in cots in the Old MCG, he was filled with both enthusiasm about the trip's progress and regret that he couldn't stay longer.

"As an anthropologist, I always think that if we just had two more months, we could get a lot more done. In a sense, this kind of work never ends. But we've uncovered an enormous amount of information. I'm altogether pleased." ■

Homecoming Plans Under Way

Plans are under way for Homecoming 1991, scheduled May 2–5. Details are incomplete, but the following information is available:

Activities for all schools

Alumni golf tournament at Jones Creek, Friday, May 3 at 10 a.m.

MCG Foundation Board of Directors meeting, Saturday, May 4, 1:30 p.m., Old Medical College, 598 Telfair Street.

President's Reception, Saturday, May 4, 3:30 p.m., Old Medical College

Allied Health Sciences

The School of Allied Health Sciences Alumni Association will host a brunch for alumni and faculty May 4 at 10 a.m. in the Alumni Center. This event will feature the presentation of the distinguished alumnus award.

The following Allied Health Sciences departments are planning workshops and/or luncheons for alumni weekend:

Associated Dental Sciences—continuing education program, Friday, May 3, 9 a.m. to 12:45 p.m. Registration: 8:30 a.m.

Medical Technology: Friday, May 3, 8 a.m. to 2 p.m.

Physical Therapy: Saturday, May 4, 8:30 a.m. to 4:30 p.m.

Physician Assistant: Luncheon at Partridge Inn Ballroom, Saturday, May 4, 12–2 p.m.

Dentistry

Class reunions for classes of 1976, 1981 and 1986, Friday, May 3, 6:30 p.m., River Boat Augusta.

Continuing education program and luncheon, Saturday, May 4 from 8:30 a.m. to 4:15 p.m. in room 1020 of the School of Dentistry. Registration: 8 a.m. Luncheon: 11:30 a.m. to 1 p.m. in the MCG Alumni Center. Guest speaker is Dr. W. Charles Blair of Blair/McGill & Co., who will discuss *"Business Aspects of Dentistry:* Controlling Office Overhead."

Graduate Studies

Distinguished alumnus award presentation and lecture, Thursday, May 2 at noon in room 108 of the Robert B. Greenblatt Library. This event will feature the presentation of the distinguished alumnus award.

Graduate studies luncheon, May 2 at 1:30 p.m. in the Murphey Building conference room.

Graduate studies fish fry, Friday, May 3 at 6 p.m. in the MCG Alumni Center.

Medicine

Dean's reception, Friday, May 3, 5–7 p.m., solarium of Old Medical College.

Alumni banquet, Friday, May

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Construction of the Medical College of Georgia's Ambulatory Care Center/Specialized Care Center spanning Harper Street is proceeding ahead of schedule and should be completed by 1992, according to Dr. Francis J. Tedesco, president of MCG.

Faculty Member Sharing Computer Know-How with Campus

David Waller

The ever-increasing role of computers in medicine has forced even the most hesitant professionals to learn how to use these sometimes intimidating devices. But while most people struggle to learn computers' most basic applications, there are those on campus who have not only mastered them but are actually breaking new ground.

Carrie DiLorenzo, assistant professor of medical illustration, was selected this fall by Apple Computers to be part of the Encyclopedia of Multimedia, a new program to demonstrate how the Macintosh computer can be used in the classroom.

Many people on campus already have equipment that they are not getting the most benefit from because they aren't aware of all the things it can do.

"It is really quite an honor," Ms. DiLorenzo said. "Some of the things on this disk are amazing and I'm really happy that we (MCG) got to be a part of it."

The disk is a compact disk that looks like one that might be used in an ordinary compact disk player. It has read-only memory, or ROM, meaning it cannot be recorded onto, but rather played only.

"What we want to do with this disk is show people around campus what can be done with various pieces of hard- and software," she said. "That way, they will know more specifically what they want when they are shopping for computer equipment."

Many people on campus already have equipment that they are not getting the most benefit from because they aren't aware of all the things it can do, Ms. DiLorenzo said. The disk will help her show people just what they can do with their computers.

The two programs on the disk, designed by MCG's Center for Clinical Anatomy, are entitled "NeuroAtlas: An Electronic Atlas of the Brain and Brainstem" and "Macrosurgical Anatomy of the Anterior Choroidal Artery." They are featured in the July/August edition of Syllabus Magazine.

Lecturers interested in how Ms. DiLorenzo can help design programs for classes or anyone who would like to see the disk may call Ms. DiLorenzo at (404) 721–6316. ■

Carrie Di Lorenzo works at computer

Ms. Singleton Named Assistant Dean

David Waller

Constance A. Singleton

Constance A. Singleton has been appointed assistant dean for business affairs in the School of Allied Health Sciences at the Medical College of Georgia.

Ms. Singleton graduated from Lucy C. Laney High School in Augusta and Augusta College with a bachelor's degree in sociology.

After graduating, Ms. Singleton worked in New York until she came to Augusta in 1970. She began her career at MCG in 1970 as an executive secretary. In 1974 she became an administrative assistant.

In 1983 Ms. Singleton was promoted to administrative assistant to the dean of the School of Allied Health Sciences.

She served in that capacity under Dr. Raymond C. Bard until his retirement in 1988. She remained administrative assistant to the dean when Dr. Biagio Vericella was appointed dean last year.

Ms. Singleton has served on various committees on campus, including the Student Financial Aid Committee. She is a member of St. Mary's Episcopal Church. ■

Dental Hygiene Program Begun in Rome

David Waller

The University System of Georgia Board of Regents has approved establishment of a three-year pilot external associate in science degree in dental hygiene to be located in Rome, Ga.

The Medical College of Georgia will direct the program, with Floyd College in Rome providing support and general education and core curriculum courses.

"This is a need which is greatly felt by the regents and we're fortunate enough to be connected with our sister institution, Floyd College," said Dr. Biagio Vericella, dean of the School of Allied Health Sciences.

The school has accepted 14 students to begin fall quarter. The faculty will consist of one full-time coordinator and one dental supervisor.

"We're hoping to better serve the dental hygiene needs to the people in north Georgia," said Gail Winkley, acting chairwoman of the Department of Associated Dental Sciences.

This is a need which is greatly felt by the regents and we're fortunate enough to be connected with our sister institution, Floyd College.

—Dr. Biagio Vericella

Students will receive an associate science degree in dental hygiene and will officially graduate from MCG.

The study of dental hygiene has not previously been offered in northwest Georgia, and Floyd College is the only institution offering these studies in that area.

"This opens up a whole new field of education for them," Ms. Winkley said. ■

School Enrolls Record Number

The School of Allied Health Sciences has a record enrollment this year of 491 students, according to Dr. Bernard J. Abbott, MCG registrar.

This compares to 454 students in fall 1989 and 398 in fall 1988—a 23.4 percent increase in the past two years. Allied health sciences applications increased 37.1 percent over 1989 figures, Dr. Abbott said.

Eight-hundred-and-twenty-five undergraduate students are enrolled in all five schools for the fall quarter at the Medical College of Georgia, 5.8 percent more than in 1989 and 18.5 percent more than in 1988, Dr. Abbott said.

Last year's fall undergraduate enrollment was 780; 696 undergraduates were enrolled in 1988.

The increase reflects the success of a new MCG recruitment program begun in 1987, according to Dr. Abbott. The program's main goal has been to bolster undergraduate enrollment, which was on the decrease in the 1980s. "This program has paid for itself many times over, and we need to continue to recruit effectively in the coming days of declining school-age population and increasing competition for students," he said.

This year's total enrollment—1,980—is a 1.7 percent increase over last year's enrollment and 7.8 percent more than in 1988. ■

Class Notes

Regina Wilkerson Leffingwell (physical therapy, '81), Arlington, Texas, is director of physical therapy at Kimberly Quality Care, specializing in home health care. Her daughter, Julia Elise, was born November 29, 1989 and weighed 7 pounds, 5 and a half ounces.

Lola Sicard-Rosenbaum (physical therapy, '86), has accepted a position as senior physical therapist at the National Institutes of Health in Bethesda, Md. In addition to clinical responsibilities, she will be involved in research conducted by the Department of Rehabilitation Medicine and support of research conducted by the 12 institutes of the NIH. ■

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Dr. Zwemer Reflects on 24 Years of Dentistry, Administration

Ingrid Heggoy

Challenge and accomplishment are important to Dr. Thomas J. Zwemer, vice president for academic affairs at the Medical College of Georgia. And he has found them throughout his career as a dentist, educator and administrator.

"The greatest achievement in dentistry since I've been involved with it is the advances made against dental caries," said Dr. Zwemer, who retires in January after 24 years at MCG. "Dentists and organized dentistry took up the cause with fervor, and they made a difference. But now people are keeping their teeth longer, and the greatest problem in dentistry is periodontal disease.

"So much money, effort and time goes into to restoring a tooth to function and good looks, and dental schools emphasize that. But now we're very good at helping people keep their teeth intact, and we need to teach them how to keep their gums healthy, so they'll keep those teeth."

Dr. Zwemer believes that organized dentistry can help.

"This is a societal issue, a professional issue. If organized dentistry took up periodontal disease with the same fervor that we took up with the prevention of dental caries, we could have the same impact."

Dr. Zwemer is a believer in organized dentistry. A member of the American Dental Association and various regional dental societies for more than 40 years, he recently was made a lifetime member of the Georgia Dental Association.

"I have always urged all my students to join organized dentistry. I believe it is extremely important, and when I worked with graduate dental students, I had my department fund their memberships. It gives them access to so much, a sense of what their profession is about, not to mention the professional journals, which give us a chance to learn," he said.

"Professional stimulation and interaction is so important, especially in dentistry, which is such a solitary profession. It is easy for a person to become isolated, to become stale, without that professional community."

Dr. Zwemer has been involved with students all his years in dentistry, and knew he wanted to teach from the time he was in high school.

The son of a building contractor and a child of the Depression, his father stressed independence to him, his brother and two sisters. Three of the four children became dentists, and the fourth works as a medical counselor at Loma Linda University in California.

"I had always wanted to teach, and my father felt we should do something in which we could be independent. Given my scientific interest and mechanical inclination, I decided dentistry was the way to accomplish what I wanted."

After graduating from the University of Illinois College of Dentistry in 1950, Dr. Zwemer accepted a position as an instructor of pediatric dentistry at Marquette University School of Dentistry. He quickly got a taste of administration when he became acting chairman of the Department of Pedodontics and then director of the Department of Oral Rehabilitation at Marquette. He then joined the faculty of Loma Linda University School of Dentistry as professor and chairman of the Department of Orthodontics.

"As it turned out, I moved quite quickly from only academic assignments to roles that included administration. I began giving academic assignments to faculty, designing curricula, writing grants," he said.

"I didn't really seek out these positions; I was invited to them. I think I accepted responsibility and delivered on it, so I received more responsibility. I moved on in this way, and accepted positions that were offered to me and were a challenge. I enjoy working in administration."

His hard-working attitude caught the attention of Dr. Judson C. Hickey, the founding dean of the MCG School of Dentistry. In 1966, Dr. Hickey invited Dr. Zwemer to come to the new dental school in Augusta as assistant dean for clinical affairs.

"The challenge and opportunity brought me here from California. Being a part of a new dental school is an exciting idea, and the chance to work with Jud Hickey was something I couldn't pass up," he said.

"We did a lot of work in a short time, starting this dental school. We really had total support from the president and the Board of Regents, and we were lucky in bringing in some great faculty at the beginning, some of whom are still here."

In 1984, he was invited to make yet another move, from administration in the dental school to vice president for academic affairs. Once again, he took the changing horizons in stride, and he still believes in the importance of academics.

"Whether I'm working with students or in administration, it's still education. The chief academic officer is dedicated to seeing that the academic community works well together, to see that they accomplish their goals and educate minds." ■

Dr. Thomas Zwemer works at office computer.

Dentist's Technique Inactivates Herpes Virus

Ingrid Heggoy

A Medical College of Georgia dentistry professor has found a way to inactivate a virus that is a common cause of cold sores with a technique that may one day be able to prevent the spread of the deadly AIDS virus through blood and blood products.

Dr. John Erbland, associate professor of oral biology and biochemistry at the MCG School of Dentistry, has in the laboratory successfully inactivated the herpes simplex virus I without damaging living cells.

Herpes simplex I causes recurrent cold sores in and around the mouth. When the virus infects a person, it sometimes migrates up the nerve and remains dormant in the spinal cord, only to reactivate and cause another cold sore in the same spot weeks or even months later.

"This is a method to inactivate the virus, not a cure," he explained. "There is no known cure for the virus."

His technique inactivates the virus by breaking down the protective envelope that surrounds it. The herpes virus, like the AIDS virus, has a thin outer membrane, or envelope, without which it cannot survive and infect living cells.

This treatment would kill the virus that is free in the blood, but the virus would still be present in already infected cells. Therefore, the treatment could be an effective preventive measure and could help to control an outbreak, but could not eradicate the virus from the body.

Dr. Erbland's research is unique in that the agents he is using, called lysophosphatidylcholines, or LPCs, are normally found in small amounts in living tissues and are less harmful to living cells than many other chemicals that inactivate the virus.

"We're looking for an agent that is the most harmful to the virus without killing the living cells," he said.

LPCs are normally found in small amounts in blood plasma and are closely related to substances normally found in the cell membrane. One kind of LPC is used by the body to make lung surfactant, which lubricates the lungs.

"Right now, we are fine-tuning the molecule, to make it the most harmful to the virus without injuring other cells," Dr. Erbland said. "We do that by varying the length and flexibility of the hydrocarbon chain, or tail of the molecule. If it's too short, it doesn't kill the virus. If it's too long and rigid, it kills the virus but can kill other cells as well. By making the tail sufficiently long and flexible, the virus can be killed without injuring the other cells. We are trying to discover the optimal length for effectiveness and safety."

The next step will be to determine whether the process works as well in a living creature as it does in the laboratory.

If so, it may be possible to use the treatment on recurrent herpes infections, according to Dr. Erbland. It also could be used to treat those who develop a systemic herpes infection, one which affects the entire body, a rare but dangerous occurrence.

"Every once in a while herpes becomes systemic, which is life-threatening, and right now there is nothing we can do about it," Dr. Erbland said. "This agent may be able to inactivate it in the body—a new lifesaving treatment."

Another possible use is to treat blood or blood products before they are given to patients to prevent AIDS infection. For example, hemophiliacs are at a higher risk for AIDS because of the blood-clotting agents they must receive regularly. These are gathered from donor blood and may carry the AIDS virus, although screening techniques greatly reduce the chance.

"If this agent was added to blood or blood fractions before being given to patients, even if the blood contained the AIDS virus, it would be inactivated before entering the body and the person wouldn't develop the disease," Dr. Erbland said.

Much work remains to be done to determine whether LPCs are as promising as they appear. Dr. Erbland plans to continue this research, in part with a $15,000 National Institutes of Health grant he received in spring 1989.

But the research with the AIDS virus or other possible applications he will leave to others in the medical field who have the materials and facilities to conduct such projects.

"I'm a dentist," he said. "And this is what I'm familiar with, which is why I began this research with herpes. The other possibilities are there for others to study.' ■

Bone-Replacing Procedure Offers New Hope for Denture Wearers

Ingrid Heggoy

A dental researcher at the Medical College of Georgia has found a way to replace bone resorbed by the lower jaw when teeth are lost.

The finding makes it possible for people to wear dentures who might have had to go toothless before and may be a treatment for those with some types of bone deformities.

"We have been looking for a better way to augment the bony ridge of the mandible (lower jawbone)," said Dr. Mohamed Sharawy, a professor of oral biology and anatomy at the MCG School of Dentistry.

"We had the idea of bone induction, or causing new bone to grow. We then had to find a way to make it grow, and make it grow in the shape needed."

In many people who lose their natural teeth, the bony ridge that holds teeth is resorbed by the body. Without this ridge, dentists can't fit the patient for dentures.

In cases like these, dentists have tried several solutions, with mixed success. They may implant into the remaining bone a metal framework with small protrusions on which dentures can be attached. But this is an involved and somewhat complicated procedure, and sometimes there is not enough bone left to use this approach, Dr. Sharawy said.

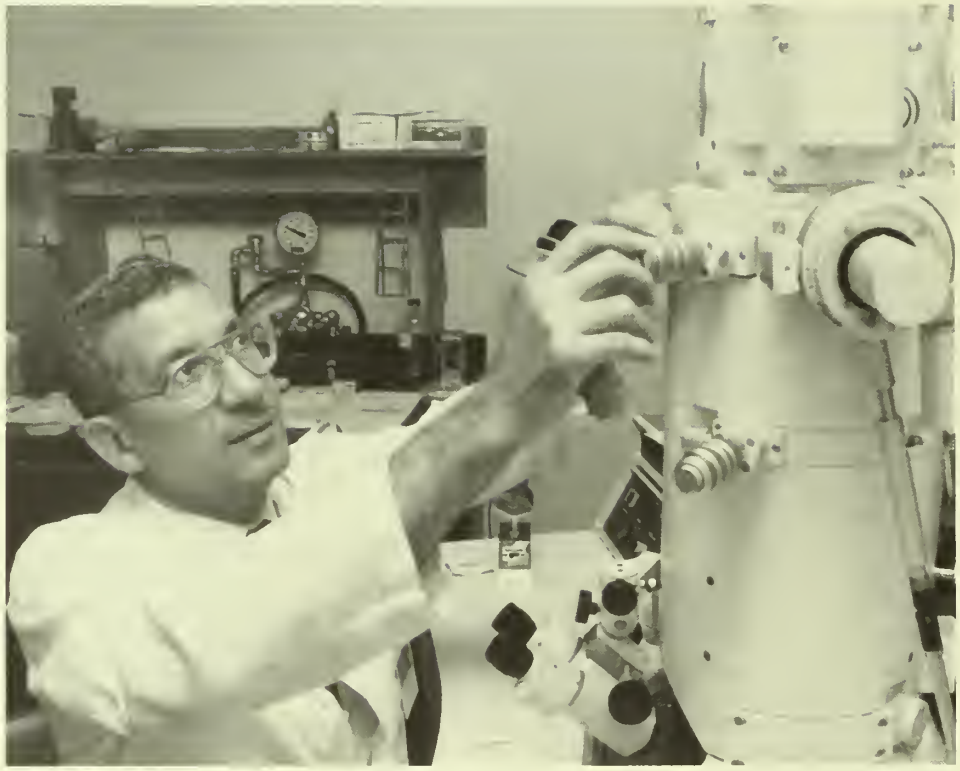
Dentists also have used bone grafts taken from the hip to replace lost bone, but the graft often is resorbed by the body, as the original ridge was, according to Dr. Sharawy.

Another technique is making a small incision in the gum and placing crushed coral on the bone to build up the ridge, but chewing places so much pressure on the material that it shifts, Dr. Sharawy said.

"We needed to get cells that were not growing bone to begin doing so," he explained. "We found that demineralized bone powder could do this."

Demineralized bone powder is made from bone retrieved from organ donors. The bone is treated and sterilized and all minerals are removed from it, leaving a small amount of bone material.

During a minor surgical procedure, Dr. Sharawy places a shaped block of a porous substance, called polysulfone—which is about as hard as bone—on what remains of the ridge. He then sprinkles it with demineral-



Dr. Mohamed Sharawy

ized bone powder. The powder causes cells called fibroblasts, which normally make collagen fibers found in connective tissues, to be transformed into bone-forming cells. The new bone grows through the small holes in the polysulfone and connects it firmly to the jawbone in 60 to 90 days.

"Bone actually grows up through the holes in the material and anchors it to the mandible," Dr. Sharawy said. "And the substance will not be absorbed by the body, so we now can actually rebuild the lost bone and fit the patient with dentures or use it for insertion of implants."

His research on augmenting the ridge is so promising that the procedure is now being used on dental patients in the U.S.

"This is a real breakthrough in that we can grow bone where we need it, in the shape we need it," Dr. Sharawy said. "It isn't difficult to grow bone as a tissue, but before it was impossible to grow it in a specific shape."

Dr. Sharawy now is looking for a way to grow useful bone for the upper jaw, a procedure which he said is much more complicated because the bone already is less dense. Also, it can't be augmented from the gum line as the lower jaw is, because there wouldn't be enough room left for dentures.

"The maxilla is much more of a problem than the mandible," he said. "We want to augment it from the sinus to build enough thickness to be able to put a metal implant in, to attach dentures to. We are doing research in which we place a small block of the substance at the bottom of the sinus cavities, just above the maxilla, and put the demineralized bone powder on it."

If the research shows the technique is practical, it too may soon be an alternative for dental patients.

This could have implications in other areas of reconstructive surgery, according to Dr. Sharawy. Bone grown with this technique could be used to correct bone deformities such as those caused by accidents or surgeries that must remove bone, such as some cancer operations.

Dr. Sharawy's previous and ongoing studies are funded by the National Institutes of Health and the Peace Fellowship Project from the Institute of International Development, a program designed by the U.S. government to aid Arab and Israeli researchers. ■

Hinman Luncheon Scheduled March 23

The 17th annual luncheon for MCG alumni at the annual Thomas P. Hinman Dental Meeting will be held March 23 from noon to 2 p.m. at the Atlanta Hyatt Regency, 265 Peachtree St. NE.

The Regency is about a block and a half from the Inforum, where the meeting will be held.

The cost of the catered luncheon, co-sponsored by the MCG School of Dentistry and the school's alumni association, is $12 with advance reservations made through the Alumni Center or $15 at the door.

The reservation deadline is March 4. Contact Shirley Taylor, alumni activities director, at (404) 721–2123 for more information. ■

Regional Dinner Slated Feburary 1

The School of Dentistry Alumni Association's Augusta regional dinner will be held Feb. 1 at the Old Medical College. The event will begin with a social hour at 7 p.m., followed by dinner at 8 p.m. All dental alumni and dental hygiene alumni are invited. For more information, contact Shirley Taylor at (404) 721–2123. ■

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Dean's Interlude

Gregory L. Eastwood, M.D.
Dean, School of Medicine

"To consider dear to me as my parents him who taught me this art... to impart to my sons (and daughters)... the precepts and the instruction."

Hippocrates
5th century B.C.

We physicians have passed on the science, the art, the traditions of our profession to our literal and figurative sons and daughters for centuries. Most of us do this, when given the opportunity, as a matter of everyday life at home and at work. Some of us who are medical school faculty actually get paid to do it!

In recent years, many physicians have become soured on the profession. Although there are ample reasons for us to be concerned about the future of medicine, and the ills of medicine must be dealt with, I have not yet heard anyone tell me that medicine no longer offers the opportunity to relieve suffering, to understand the human condition in a manner that is available to few, and to lead a fulfilling and useful life. Commonly, I have heard colleagues tell me that they have advised their children against considering medicine as a career or, in my experience interviewing medical school applicants, had them tell me that they have decided to apply against the advice of their physician parent. (Good for them!)

The nadir in number of applicants to U.S. medical schools seems to have passed. For the class that entered this past summer, nationwide the applicant pool had increased about 8 percent. At MCG, the number of applicants to the School of Medicine had increased over 15 percent. And so far this fall at MCG, applications for next year's entering class are ahead of last year's. Perhaps young men and women are not heeding the discouraging remarks of their elders or, more and more, we elders are encouraging them to enter medicine and help us deal more effectively with some issues that threaten our profession.

One of the symbols of the passing of the science, art and tradition of medicine to those who follow us is the conferring of the hood at the time of graduation from medical school. The hooding ceremony at MCG has become a very meaningful experience for the graduates and their loved ones. Although the general university-wide graduation exercises occur the next day, the hooding ceremony is regarded by many medical students and their families as the high point of graduation.

This past June we added something to the hooding ceremony that we anticipate will be a tradition for many years. During the actual act of hooding, in which the dean places the hood upon each graduate in succession, those graduates whose parents were physicians were asked to have their physician parents assist me in placing the hood over the head and shoulders of their son or daughter. The smiles, the tears, the hugs and the appreciative comments were evidence of the great meaning this simple act had for those new physicians and their parents, and perhaps are testimony to the meaning to all of us of imparting to our literal and figurative sons and daughters "the precepts and the instruction" of our profession. ■

Magazine Honors Dr. Hames

Dr. Curtis G. Hames ('44), a family practitioner in Claxton, Ga., has been granted an award for distinguished achievement by Modern Medicine.

Recipients were selected by the editorial board of the Cleveland-based magazine, a national clinical journal for primary-care physicians.

"These awards honor clinicians, educators and researchers whose important contributions have advanced the practice of medicine," said Martin Stevenson, editor of Modern Medicine.

The magazine noted Dr. Hames' contributions to the understanding of genetics, immunology, cancer, hypertension, stroke, myocardial infarction, pesticide pollution, neurohormones and psychosocial determinants of disease. Dr. Hames currently is helping establish a national network of primary care research centers that will eventually be linked to a world network, pushing for fundamental changes in the character of medical education and developing a primary-care clinic and research facility in Nigeria.

"I'd like to see (medicine) return to being a labor of love and a communion with patients," Dr. Hames was quoted in the article.

Other honorees are Dr. Clifton R. Cleaveland, a general internist in Chattanooga, Tenn.; Dr. Aram V. Chobanian, dean of the Boston University School of Medicine; and Dr. C. Everett Koop, former U.S. surgeon general and present chairman of the National Museum of Health and Medicine Foundation and the National 'Safe Kids' campaign in Washington, D.C. The four recipients were honored in the June 1990 edition of Modern Medicine. ■

Dr. Silver Named Academy V.P.

Dr. William E. Silver, a graduate of the Medical College of Georgia School of Medicine and a facial plastic surgeon in Dunwoody, Ga., has been named southern regional vice president of the American Academy of Facial Plastic and Reconstructive Surgery.

He was elected during the academy's 1990 fall meeting in San Diego. The academy is the world's largest association of facial plastic surgeons. Members must be board-certified surgeons with training and experience in facial plastic surgery. Most of its fellows are also fellows of the American College of Surgeons.

Dr. Silver is immediate past vice president of the American Academy of Otolaryngology—Head and Neck Surgery. He also is chairman of continuing medical education for the Greater Atlanta Otolaryngology—Head and Neck Surgery Society and the Georgia Society of Otolaryngology—Head and Neck Surgery. He is one of 34 preceptors for the American Academy of Facial Plastic and Reconstructive Surgery and is on the teaching staff of the Emory University Department of Otolaryngology. ■

MCG Alums Battle Diseases in Thailand

Toni Baker

Fast-food chains dot the Thailand skyline.

And the westernization of this southeast Asian country means atherosclerosis is becoming more visible as well.

But Drs. Vijitr Boonpucknavig and Somnate Boonpucknavig need look no farther than the ponds and lakes of their country to find major killers.

Here the citizens fish, then clean, spice and eat their catches raw.

Some of the fish contain a deadly, aggressive parasite—the liver fluke, opisthorchis viverrini—that attacks the liver, causing inflammation and sometimes perforation of the bile ducts. The liver also sometimes develops cancer.

But it doesn't stop here. Parasites in the liver can shoot out an antigen that damages the kidneys. A drug is available to kill the parasites but it's expensive and has many side effects. Cooking the fish also will kill the parasites, but public education can be difficult, especially in the jungles and forests of Thailand. Dr. Vijitr, professor and chairman of the Department of Pathology at Ramathibodi Hospital in Bangkok and his wife, Dr. Somnate, professor and chairwoman of the Department of Pathobiology at Mahidol University, have pooled their specialties of pathology and immunopathology to wage war on these parasites and others in their homeland where infectious diseases are the number-one overall killer.

Both doctors did research fellowships at MCG in the late 1960s. October found them back at MCG for a Department of Pathology Alumni Reunion where they talked about the diseases they battle.

That takes us back to the ponds and lakes of Thailand, because it's also here that mosquitoes breed that bite people and spread malaria. Malaria is a protozoa that affects red blood cells, Dr. Vijitr explained. Wherever the cells go, which is to every major organ in the body, the malaria parasite can do damage.

Drs. Vijitr and Somnate are trying to understand the damage that can be done by this infectious agent. "If you know the mechanism of damage, you can try to prevent it," Dr. Vijitr said.

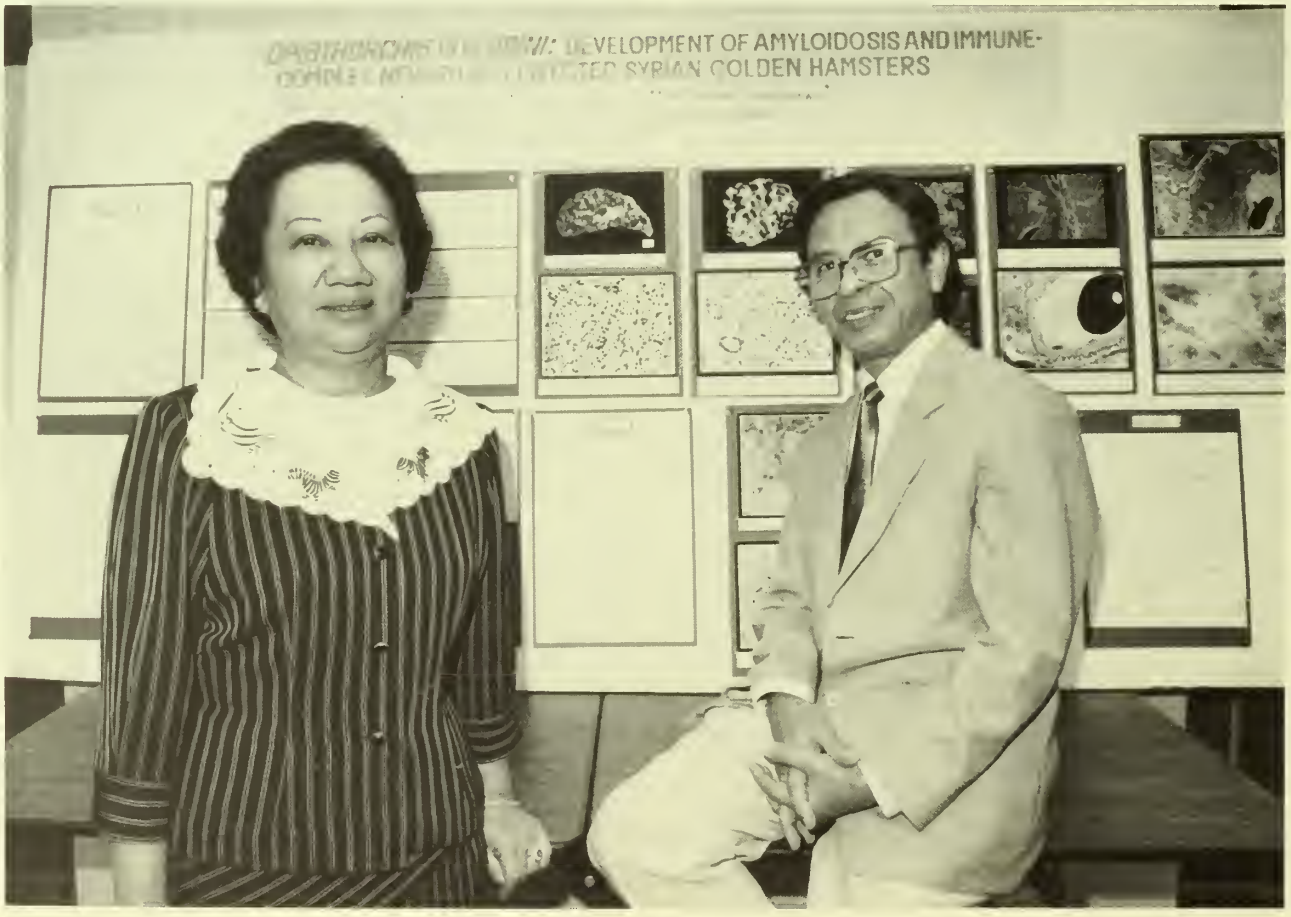
A resurgence of malaria occurred in Thailand after the Vietnam War and the parasite itself has changed as well as the natural history of the disease.

They are the same mosquitoes and same parasites, but they seem to be doing more harm. For example, 15 years ago, doctors first began to see pulmonary edema in malaria victims. The kidneys had been and are the most affected by malaria. The brain also is affected. So far, the heart is spared.

Vaccines can be useful in battling the parasites, but because they move through various stages while in the body, vaccinations have to be given for all different stages.

Drs. Vijitr and Somnate have been working with the U.S. Navy for about five years to develop a more effective vaccine. He's also working with the World Health Organization on a textbook on malaria.

There already is a book full of drugs, led by quinine, that can help in battle the parasites, but even combinations of the drugs still are not 100 percent



Drs. Somnate and Vijitr Boonpucknavig

effective. So Drs. Vijitr and Somnate also are helping look for new drugs.

"You need to better understand the changes the parasites go through in the body and see what drugs exist or are needed to kill the parasites in the various stages," he said. ■

Alum Refining Sharpshooter for Cancer Cells

Toni Baker

Dr. David H. Munn wants to improve the aim of the body's immune system.

He wants to make this natural defense system a sharpshooter for cancer.

"The issue here is how can we use the exquisite selectivity of the immune system to target cancer cells," said Dr. Munn, a 1984 graduate of the Medical College of Georgia School of Medicine who is now back at MCG as a cancer researcher and pediatric hematologist/oncologist.

"The immune system is very good. It has access to every single cell in the body," Dr. Munn said. "It can make physical contact with every cell and check to see whether it is infected with a virus, whether it has a bacteria growing in it or around it. That's its job. That's what it does 24 hours a day. That's why blood is sterile. The immune system is constantly removing any germ that gets in it."

Some doctors—including Dr. Munn—suspect that the immune system also battles cancer cells.

It's called the immune surveillance theory and it says that cancerous cells may arise in the body all the time but that the immune system gets rid of most of them.

But once in a while, a cell will sneak past the surveillance.

"The only tumors that are successful, the only ones that are able to grow and cause a problem, are the ones that don't look so foreign that the immune system automatically rejects them," Dr. Munn said.

"In a sense there is a selection process that goes on even before a tumor is able to get past the single-cell stage. So it must be well disguised."

Dr. Munn wants to capitalize on the ability of the immune system to battle cancer by selectively revving up that capability when a child or adult gets cancer.

"The question is not just how can we turn on the immune system. But how can we turn it on in a selective way that causes harm to the cancer and doesn't cause unacceptable harm to the normal cells of the body," the researcher said.

A cell called a macrophage may be able to offer the selectivity needed.

Macrophages are probably involved in helping tumors grow. They participate in the production of new blood vessels and fibrous tissue and all the things a tumor needs to grow and thrive.

Macrophages also appear able to distinguish between a normal cell and a cancer cell.

And, put these macrophages in a test tube and, under the right conditions, they can even be made to see tumors as the enemy.

What Dr. Munn wants to do is create a test tube environment for the macrophage which is like its environment in the body's immune system. "For the first time, in the last three or four years, we have the array of purified, well-characterized hormones and growth factors that allow us to put together a recipe that comes close to exactly duplicating the environment in the body," he said.

When the recipe is right, he can alter the levels of the different components of the immune system and look to see which combination makes the largest number of macrophages that are the most aggressive toward tumors.

Different combinations might be needed to fight the many different cancers that affect children and adults.

Getting the ingredients these days is no problem. Recombinant DNA technology makes it possible to make buckets full of some 20 different factors of the immune system.

These factors are proteins that are secreted by the immune system or other cells in the body. They act on the cells of the immune system and are the body's regulatory mechanism for keeping the immune system up or down.

Dr. Munn is trying to put these factors together in a fashion that provides a laboratory model for what is actually going on in the tumor environment.

Different components could be altered in this test-tube setting to see what would influence the immune system to aggressively fight a particular cancer.

"We need a laboratory experiment that will let us take the hundred combinations that would be possible with the different agents and say, 'Let's try this one because this one works in the test tube and that test tube is a good model for what is going on in the body,'" Dr. Munn said.

Such a model would mean new cancer treatments would not always have to be proven or disproven by trial and error on humans. ■

Class Notes

Dr. William C. Lindsay, Knoxville, Tenn., has been elected to fellowship in the American College of Cardiology. He is a cardiac electrophysiologist for East Tennessee Heart Consultants.

Dr. Powers Peterson has been named director of the New York Hospital-Cornell Medical Center Laboratory of Clinical Hematology.

Dr. H. Wilder Smith ('46), Swainsboro, is doing primary care medicine and opened a museum and memorabilia for viewing on August 3, 1990. This was a one-day viewing but more will be held in the future.

Dr. Dan Hanks Jr. ('69), Rome, Ga., has been named president of the Georgia Radiological Society.

Dr. Hugh M. Gloster Jr. ('89), Atlanta, began a three-year residency at the Emory University Hospital in July 1990. The residency will lead to specialization in dermatology.

Navy Lt. Jerry W. Hamlin ('90) has completed the Officer Indoctrination School at the Naval Education and Training Center in Newport, R.I. ■

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All past presidents of the SMAA are lifetime members of the board.

Homecoming... *cont. from page 13*
3, 7 p.m., ballroom of Old Medical College. This event will feature presentation of the distinguished alumnus award.

School of Medicine Alumni Association Board of Directors meeting, Saturday, May 4, 8:30 a.m., Old Medical College.

School of Medicine Alumni Association luncheon, Saturday, May 4, noon, Old Medical College.

Class reunions for the classes of 1941, 1946, 1951, 1956, 1961, 1966, 1971, 1976, 1981 and 1986 and a dinner for alumni who are not having a reunion will be held Saturday, May 4 at 7 p.m. Locations will be announced.

Emeritus Club luncheon, Sunday, May 5, 11:30 a.m., ballroom of Old Medical College.

Nursing

Nursing program and dinner, Thursday, May 2, 7 p.m., Old Medical College. Dr. Vickie Lambert, dean of the School of Nursing, will be guest speaker, discussing *"MCG Nursing: The New Decade."* This event also will feature the presentation of the E. Louise Grant and Phoebe Kandel Rohrer Founders awards. ■

Research Symposium Slated May 17–18

The Medical College of Georgia School of Graduate Studies will sponsor the first annual University System Symposium May 17–18 on the MCG campus.

The symposiums will draw scientists throughout Georgia, especially those affiliated with university-system institutions, to update each other on their research and explore possible collaborations, according to Dr. Lowell B. Greenbaum, dean of graduate studies and vice president for research at MCG.

The theme of this year's symposium is "Biomedical Research Brings A Better Quality of Life." The guest speaker will be Dr. Stanley Cohen, a faculty member at Vanderbilt University and a Nobel prize winner for his work on epidermal growth factor.

The symposium also will feature research poster presentations and panel discussions. Scientists throughout Georgia are invited. ■

Nursing Dinners Slated

The School of Nursing Alumni Association's Augusta regional dinner will be held March 15 at the Old Medical College. The Atlanta regional dinner will be held April 26 at the Sheraton Century Center, 2000 Century Blvd. NE in Atlanta. For more information about either, contact Shirley Taylor at (404) 721–2123. Watch for more news about the School of Nursing in its next edition of *AlumNews*, which will be published in the spring. ■

AlumNews

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Writers Toni Baker
Christine Hurley Deriso
Ingrid Heggoy
David Waller

Design Tina Frix
Photography Phil Jones

Deadline for submitting information for publication in the spring issue of Alumnews is Feb. 19, 1991

Alumni! Let us know what's new with you by taking a moment to fill out this form. Also, please send us your curriculum vitae so we can keep your files up-to-date.

Today's date

Name

Phone

School graduated from

Degree Class year

Street address

City State Zip

❑ Check if new address

Present specialty and place of practice or training

Professional news

Personal news (b&w photos welcome)

Please send to : Christine Deriso; Alumni Center FI-100; Medical College of Georgia; Augusta, GA 30912

Health-Care Express

The mission of the Medical College of Georgia is to help all the people of Georgia, but for those at an economical disadvantage, a trip to Augusta poses a real hardship and in some cases is impossible.

To help remedy that problem, Dr. Virgil McKie, associate professor of pediatric hematology/oncology at MCG, started a series of satellite pediatric sickle cell clinics across the state in 1983.

"Many of these people can't afford the kind of specialized health care they need in their area," Dr. McKie said. "And they certainly can't afford to come to Augusta for routine care."

The first of the clinics was established in Waycross with five patients in 1983. Since then, additional clinics have been added in Brunswick, Savannah, Valdosta, Albany and Dublin. More than 800 patients are now followed in the clinics.

"It's important that this disease be diagnosed early," Dr. McKie said. "We are very fortunate to have an effective newborn screening program in place which allows for early detection and rapid assignment to a satellite clinic near the baby's hometown."

Some patients are diagnosed by their family doctor and referred to the clinics, while others are diagnosed in the clinics.

Educating the patients and their families is an important part of the satellite clinics.

"We teach them how to eat and what sorts of activities may result in pain crises," Dr. McKie said. "We also teach them how to access the health care systems that may already be in place in their area."

Education doesn't end with the family and patients, according to Dr. McKie. Educating local health department officials as well as family doctors and nurses is also a mission of the satellite clinics. Dr. McKie admits that he too has learned a lot as a result of the clinics.

"It has been very educational for me to see the resources across the state of Georgia," he said. "Georgia has a real problem with sickle cell disease and being able to evaluate and hopefully improve the system is very important to me."

The satellite clinics have helped MCG in other ways as well. It has allowed researchers to study patients with the disease and explore better ways of treating sickle cell patients, according to Dr. McKie.

The result of the clinics has been a significant decrease in the mortality and morbidity rates of pediatric sickle cell patients, according to Dr. McKie.

And even though MCG can take much of the credit for that, Dr. McKie said that ultimately, the patient's health depends very much on how the patient takes care of himself.

"Occasionally it becomes necessary for patients to come to Augusta for various procedures and treatments, but most of it can be done in their hometowns," Dr. McKie said. "It's a good system."

The number of pediatric sickle cell patients is growing, but that is good because it means that these patients are living longer and better lives, according to Dr. McKie.

"It's a lot of work. The work at home doesn't stop when I'm gone, and I have to catch up when I get back," Dr. McKie said. "But it is well worth it."

—David Waller

This is not a story about sickle cell disease even though it can't be told without talking about it. It isn't fiction, even though nothing more heart-warming could have been made up. And it certainly isn't written by a Pulitzer Prize-winning journalist, even though no one has ever written with more admiration for his subjects than I have.

The characters in this story are very simple. They aren't world leaders or undercover agents, but rather a 5-year-old girl named Cocoa Winfry, her mother and a very special friend who also happens to be a doctor.

"When we moved to Augusta, I had very little experience with sickle cell disease," said Dr. Kathleen McKie, an MCG pediatrician. "That is just where the job opening was and so I took it."

Dr. McKie is no stranger to health care. She is a third-generation doctor, the second to be a pediatrician and is married to Dr. Virgil McKie, MCG pediatric hematologist/oncologist.

Dr. McKie took that job nine years ago and has since, by anyone's standards, become very much an expert on sickle cell disease.

Cocoa has been under Dr. McKie's care since birth, but Cocoa was in the clinic even before she was born.

"Cocoa has a brother who also has sickle cell and I brought him in to see Dr. McKie while I was pregnant with Cocoa," said Patricia Winfry, Cocoa's mother.

Since Cocoa is the second child in the family with the disease, they had already adjusted to the special needs of a child with sickle cell.

Sickle cell is a disease that not only must be dealt with on a physical level, but also psychologically. Teaching the children and their parents how to deal with it is an important part of what Dr. McKie does.

"There are two major things we focus on. One is the physical well-being of our

Tender Loving Care

patients. But probably almost as important is their self-esteem, how they see themselves and what they can do for themselves," Dr. McKie said.

Sickle cell can be a debilitating disease and too often children are inclined to become "wimpy," according to Dr. McKie. "We really work toward teaching our kids to be tough and to realize that they can do it and that they can be what they want to be," she said.

Dr. McKie admits there are certain things people with sickle cell disease cannot do. Another part of her job is helping the children realize what their limitations are and how they can live with those limitations.

"This past summer I had to constantly explain to Cocoa that she had to take it easy in the heat," Ms. Winfry said. "We have to make her sit down every once in a while, because she will just push herself to the extreme."

Dr. McKie compares herself to a second mother for many of her patients. Sometimes she has to scold her children and sometimes she praises them.

As in most parent-child relationships, getting the children to eat right is a constant struggle for Dr. McKie. In sickle cell patients, proper nutrition is even more important than in other children.

"As the children get older, we talk to them about what good nutrition is. Some of our kids don't eat right and they get away with it, but we want them all to know what they are supposed to do, because some of them don't get away with it," Dr. McKie said.

Sometimes activities that most parents and children would consider harmless can cause trouble for the sickle cell patient. Dr. McKie recalls an example of a little boy who went to Six Flags amusement park and got too hot. He suffered the worst pain crisis of his life.

Another child was playing with a video game set that he had gotten for Christmas and a pain crisis involving his hand resulted.

Pain crises occur when the blood cells are unable to get oxygen to certain parts of the body because of the cells' abnormal shape. This lack of adequate blood flow results in pain. The pain during these crises can be excruciating in one specific part of the body or throughout.

With Cocoa, these pain crises have resulted in dozens of trips to the hospital. The pain usually begins in the abdomen and hips. Over the past five years, Cocoa has had to have her spleen, gall bladder, tonsils and adenoids removed. "That's a lot of time in the hospital in five short years," Dr. McKie said.

"It is hard to explain to kids sometimes that this kind of simple and normal behavior can result in a crisis. It seems really unfair for a child to have to suffer because he played with his new toy too much," Dr. McKie said.

On the other hand, sometimes children will learn that they can manipulate people and situations by taking advantage of their illness, Dr. McKie said.

"They can tell that people are very sensitive to their being sick, and they will work it to death," Dr. McKie said. "We've got children who can miss several weeks of school for something that is not really there."

Distinguishing real problems from imaginary ones is just one of the psychological games that is played with pediatric sickle cell patients. Sometimes children have to be convinced that they can do certain things that they may be scared of.

"It is a delicate balance of pushing a child to do what he needs to do, and pulling a child away from things that he can't do," Dr. McKie said.

Dr. McKie has two children of her own. She said that dealing with the children in the clinic has helped her with her own children and vice versa. Many of her patients are the children of single parents and she is getting a taste of that now as well. He husband is a member of the U.S. Army Reserve and was recently called to active duty at Fort Stewart near Savannah.

Even though these children have a very serious disease, they are still children and have to be treated that way. Having a chance to be a child is a privilege that most take for granted, but it is one that can also be very easily taken away by sickle cell anemia.

It seemed strange to be talking to such a small child about such a serious subject. There were constant reminders that Cocoa's life is a delicate balancing act between that of an adult and that of a child. For example, I had to stop her coloring so she could list all the medications she was taking and explain how they were helping her.

"We tell our parents to expect a long, good life, punctuated by episodes that we can take care of. They know they can call any time of the day or night and working together we can keep these kids generally healthy and happy," Dr. McKie said.

—David Waller

Growing Stronger

Dr. Nazir A. Bhatti drew an ace.

He was faced with several sickle cell patients whose disease already had destroyed one hip joint and was threatening the other.

From his orthopedic surgery hand he pulled a procedure called core decompression.

He thought it might work.

Core decompression has worked for many other conditions that affect the vital juncture where the head of the thigh bone fits smoothly and snuggly into the socket of the hip bone.

Steroids taken for rheumatoid arthritis or lupus, Gaucher's disease which causes fat to circulate in the bloodstream, the bends, trauma and alcoholism all can cause bone death or osteonecrosis.

Osteonecrosis causes bone pressure and pain.

Core decompression is a way to relieve that pressure and pain while removing dead bone and making room for new blood supply and bone growth.

Later Dr. Bhatti would learn that others in his profession also would occasionally turn to core decompression for sickle cell patients when bone death was caught early.

Five years and a dozen procedures later, Dr. Bhatti has the patients to show he and others played their hand well.

"All of them have become totally pain free. They are fully ambulatory regarding that particular hip and none of them have shown any clinical or radiological signs of progression of their disease. It's very, very encouraging."

Across the board, about 10 percent of sickle cell patients get some osteonecrosis of the hip, said Dr. Paul Milner, physician in charge of the Adult Sickle Cell Clinic at the Medical College of Georgia.

Patients at MCG who've needed core decompression have been young, ranging in age from 19 to 38.

That morning, Dr. Milner had a 31-year-old man come in with pain in both hips. As part of a routine health check on these patients, Dr. Milner had an X-ray taken of the hip in March and again that day in late October. But conventional X-ray alone is not always able to pick up the subtle early changes of bone death. The dead bone shadow and live bone shadow aren't necessarily distinguishable, especially early in the process. And, by the time the X-ray plainly shows destruction, it's often too late for core decompression to help. So Dr. Milner has scheduled the patient for magnetic resonance imaging, a tool he and Dr. Bhatti agree is very sensitive in picking up early osteonecrosis.

But the MRI won't really be the final factor in what to do for this man's hip.

"We can't predict from the MRI what the clinical progress will be," Dr. Milner said. "Some of these sickle cell hips will heal on their own." And no test will really differentiate between which hip will heal and which hip won't.

Pain—the aching, throbbing pain resulting from the pressure resulting from continued bone death—is the real clue.

"Although you need the MRI for diagnosis, you don't treat MRI pictures or X-ray pictures, you treat pain," Dr. Milner said. "They ache getting in and out of the car. They ache if they walk around the mall for more than a half hour. They ache when they move in bed."

The outstanding results from core decompression at MCG may be due to the careful check kept on these patients, Dr. Bhatti said.

If pain in the hip persists for three months, the procedure is performed in which a hole about the size of the small finger is made near the top of the thigh bone. A tiny tunnel is dug slightly upward toward the head of the femur, where Dr. Bhatti removes some of the dead bone, helps relieve pressure and thus pain, and leaves an area conducive to new blood supply and bone growth. The tunnel is tiny enough not to structurally weaken the femoral head. The procedure takes about one hour. It takes the patient another six weeks to three months to heal.

"There are a lot of theories about why the bone dies in that area of the body," Dr. Bhatti said. "One of the theories put forth is that the blood flow is sluggish in that area because of the inherent anatomy of the vessels.

"In sickle cell disease, blood cells group together, coalesce, and they block those arteries. In other diseases (like Gaucher's disease), fat globules do the same thing. They block those small arteries and since the blood flow is very slow, it's very easy for this phenomenon to happen there. Once the blood flow is cut off to the bone, it dies," Dr. Bhatti said.

These bad things can happen for good reasons, because like all other organs in the body, bone is an active, dynamic, viable organ. Even the oldest person's bone is not a dead structure. It is constantly remodeling and rebuilding. Cells called osteoclasts take away dead bone and cells called osteoblasts bring new bone. That is why a broken bone heals, perhaps more slowly in older people, but it does heal.

Core decompression seems to help the bone do just that.

"There are some doubting Thomases about this procedure," Dr. Bhatti said. "Their rationale is that the majority of sickle cell hip disease does not progress. We do not feel this is an accurate assessment of this procedure."

Between magnetic resonance imaging studies, further studies in MCG's Metabolic Bone Disease Laboratory to document bone death and keeping a close check on the pain of these patients, Drs. Milner and Bhatti agree that there is an obligation to find the patients who will benefit from this procedure and to do it.

So does Dr. Tim Young, MCG orthopedic surgeon specializing in reconstruction.

"You've got to do (the core decompression) in the early stages, before you get the collapse and the incongruities that develop in the femoral head. You want to get it while it's still confined in the (femoral) head. It's worth a try," Dr. Young said of core decompression. "It's a small price to pay to potentially keep the disease from progressing."

Because when bone death progresses, the head of the femur begins to lose its smooth, rounded shape. Then it begins to work like sandpaper, damaging the smooth cup of the acetabulum where it fits.

At this point, the only real

option is total hip replacement.

Drs. Young and Bhatti agree that the total hip replacement is an important tool for treating bone disease of the femoral head.

But they also agree that, especially in these young patients who seem most affected by aggressive osteonecrosis, it's so much better to help them keep their own hips.

"The sickle cell patients do not have a great track record with the total hip replacement because of the other associated problems they have," Dr. Young said. "They are at risk for loosening and infection.

"Early results may make you think you are doing an outstanding job. But long term, the failure rate—and this is significant—approaches 50 percent at five to six years after the procedure," he said. Total hip replacements for other reasons, like arthritis, have a failure rate of less than 5 percent at five years.

If the hip has to be revised in these patients, the result of the second procedure has an even poorer long-term result.

"The worst that can happen, and this is bad enough, is that we might have to remove the components because of infection, if the infection cannot be controlled with the components in. This leaves a gap here so that bone is articulating on scar tissue and bone," Dr. Young said.

This option, called a Girdlestone procedure, means the leg pistons up and down several inches as the patient walks and that typically the patient must use crutches or a walker.

Dr. Bhatti concurs that some patients can have hip osteonecrosis over months or even years that will not progress to total hip destruction and the need for such extreme procedures.

But those patients are the minority.

"If this process is caught very early in the game and is confirmed with various tests like MRI, in our judgment, it is a very worthwhile procedure and should be attempted," Dr. Bhatti said.

"We cannot say with 100 percent predictability that if we had not operated on these patients, that they might not have progressed. But, overall, our assessment is that the majority of these patients would have progressed to hip destruction."

—Toni Baker

A Boost to Good Health

Dr. Paul Milner has dramatically improved the lives of six adult sickle cell anemia patients he treats at the Medical College of Georgia.

They were part of a pilot study in conjunction with four other medical centers throughout the country testing a new treatment, a drug called hydroxyurea, which increases the body's production of fetal hemoglobin in sickle cell anemia patients. The pilot study was funded through a three-year, $50,000 National Institutes of Health grant. Fetal hemoglobin is as effective as regular adult-type hemoglobin in the blood, and sickle cell anemia patients have a defect of only the adult hemoglobin. Therefore, higher levels of fetal hemoglobin reduce the symptoms of the disease.

"The objectives of the pilot study were, first, to see if we could raise the fetal hemoglobin level sufficiently to make these people better; secondly, to see if we could do this without drug toxicity; and thirdly, to find the minimum non-toxic dose that would increase fetal hemoglobin to a respectable level, to about 20 percent," Dr. Milner explained.

The results of this preliminary study are an unqualified success.

"All of our patients responded," Dr. Milner said. "They were all considerably helped, and it kept them out of the hospital. They all felt better, and it helped them put on a little weight, which is a common problem for people with sickle cell anemia."

None of Dr. Milner's patients have had any toxic side effects from the drug, which they have now been on three years, although it did take some time to find the correct dosage for each one. All of the patients plan to remain on the treatment.

"They still have some aches and pains, and they may need some medication for pain now and again, but this treatment has kept them from having pain crises and kept them out of the hospital, a major problem for them before," Dr. Milner said.

Dr. Milner found that the drug was effective in raising fetal hemoglobin no matter which of the five major chromosome types of sickle cell disease the patient has (see adjoining story) In all of the patients, fetal hemoglobin was raised to between 20 percent and 30 percent.

Researchers and physicians aren't sure exactly how hydroxurea works, just that it does. It seems to in some way select out the cells in the bone marrow that make more fetal hemoglobin, which is found in small amounts in the body naturally. The drug somehow allows these cells to survive better, while it interferes with replication in those cells that make the malformed sickle hemoglobin, according to Dr. Milner.

Johns Hopkins Medical Center in Baltimore, Md., has submitted a proposal to the NIH for a larger study of the drug's effectiveness. If funded, MCG will be a part of this larger study, scheduled to begin in April, with more than 30 patients receiving hydroxurea. MCG's role in this proposed study would be funded for five years by a $500,000 grant, according to Dr. Milner. ■

—Ingrid Heggoy

Scientists have long known that sickle cell anemia is caused by a gene defect and that those with less severe symptoms have more fetal hemoglobin in their blood. But they didn't know why. Until now.

Drs. Kenneth Lanclos, associate professor in the Medical College of Georgia Department of Cell and Molecular Biology, and Titus Huisman, Regents professor and chairman of the department, have discovered why.

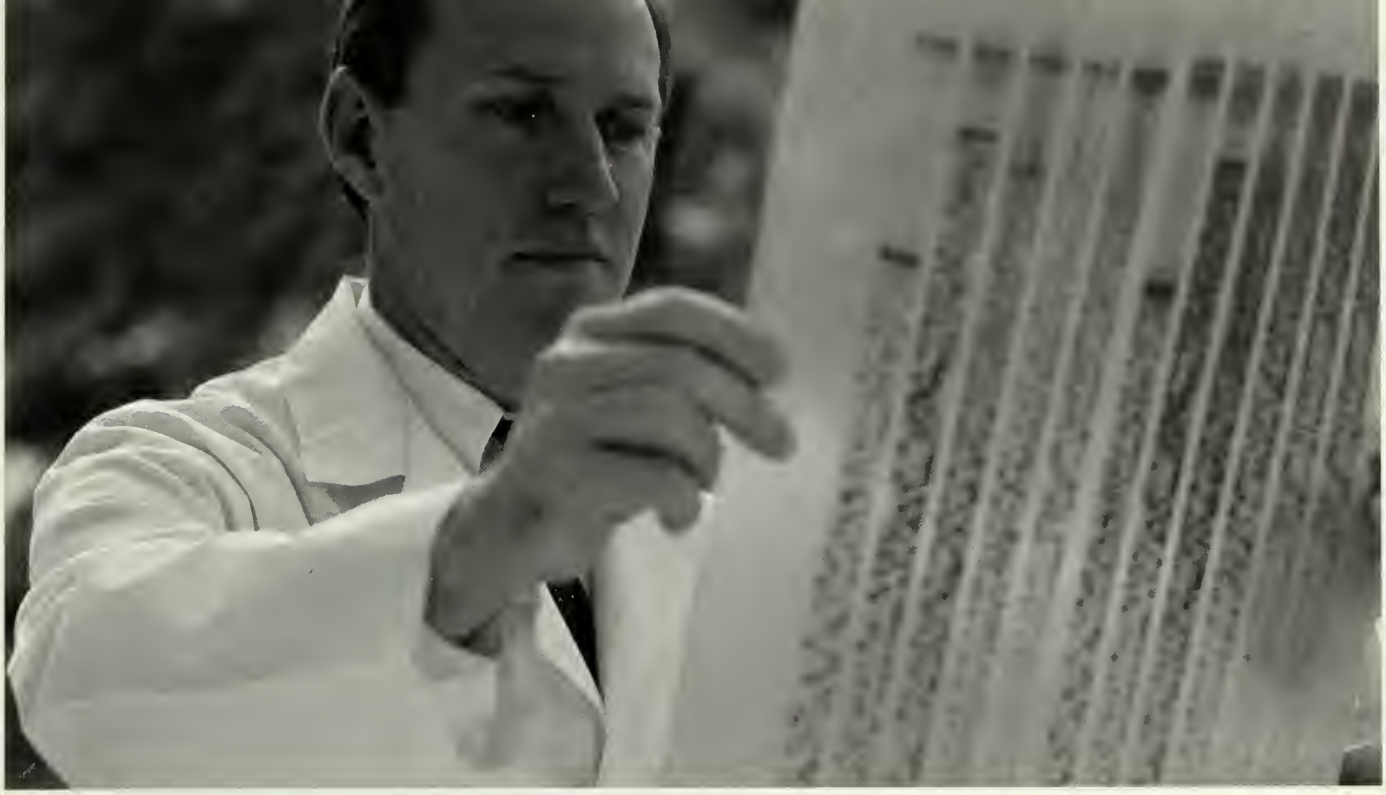
Hemoglobin is the oxygen-carrying component of the blood that travels through the body in red blood cells. In sickle cell anemia patients, hemoglobin is malformed. However, the body can make some amounts of fetal hemoglobin, the type found in the body before birth, which is very similar to adult-type hemoglobin but not affected by sickle cell disease.

Until shortly before birth, all hemoglobin in the body is the fetal type. Then, the body shuts off production of fetal hemoglobin and begins producing adult-type hemoglobin, although a small amount of fetal hemoglobin continues to be produced throughout one's life, about 1 percent of total hemoglobin.

However, some sickle cell patients produce 20 percent to 30 percent fetal hemoglobin. Since blood cells containing fetal hemoglobin don't sickle, these patients have less severe symptoms than those with lower fetal hemoglobin production.

Dr. Lanclos and his research team have discovered specific differences on the five major chromosome types which are associated with sickle cell anemia, which may help explain the variations in fetal hemoglobin production.

"We're really looking at two things," Dr. Lanclos said. "We're looking at what regulates gene expression—in other words, what allows some people to begin making fetal hemoglobin again. We're also studying the different chromo-



Keys to Chromosomes

some types sickle cell patients may have, to see what differences exist on this level.

"We've known that sickle cell patients with high levels of fetal hemoglobin have less severe symptoms than those with low fetal hemoglobin," he said. "We have been searching for the genetic difference that allows these people to have high fetal hemoglobin, and therefore be less affected."

The chromosomal differences Dr. Lanclos and his research team found are in the regulators for chromosome 11, the chromosome responsible for making the defective part of adult hemoglobin. Of the five major chromosome types they found, three instruct the body to produce low amounts of fetal hemoglobin and two direct it to produce high fetal hemoglobin. Low fetal hemoglobin is much more common, according to Dr. Lanclos.

"Sickle cell patients with low fetal hemoglobin seem to have a defect in the locus control region of chromosome 11," he said. "These people are unable to increase fetal hemoglobin production, as some other sickle cell patients and people without the disease could do if needed. They really have two different mutations on the same chromosome—the one that causes sickle cell disease and the one that prevents increased fetal hemoglobin production."

Adult hemoglobin is made up of two alpha and two beta molecular chains. Sickle cell anemia is caused by a defect in the gene on chromosome 11 that is responsible for making the beta portions of hemoglobin. The defect causes hemoglobin molecules in the red blood cells to bond with one another when they are low on oxygen, causing the deformed red blood cell. Fetal hemoglobin contains two alpha chains, as in the adult hemoglobin, but the two beta chains are replaced by two gamma chains, which do not contain the defect.

When fetal hemoglobin and adult hemoglobin are mixed in a red blood cell, the fetal hemoglobin prevents the adult hemoglobin from bonding, and so prevents the cell from sickling.

It is important to control cell sickling in patients to reduce symptoms, which include severe pain crises, during which the patient may be hospitalized for several days, and severe anemia. Sickle cells don't survive in the body as long as regularly shaped cells, so the bone marrow sometimes can't keep up with demand. If the red blood cell count drops and the patient becomes anemic, he may later have complications such as kidney problems.

The research team has studied blood samples and chromosome types from sickle cell patients from 12 countries throughout the world that are representative of where sickle cell disease is found. Countries include the United States, Canada, Turkey, Syria, Tunisia, Greece, Italy, India, Surinam, South Africa, Kenya and Nigeria. The disease is found in people from warmer climates throughout the world and has spread geographically as people migrate. People from different regions of the world often have different types of the disease, although there has been a lot of intermingling through the years.

Dr. Lanclos believes his research sheds light on several future treatment options for sickle cell anemia.

"These discoveries about the mutation in the controlling region of chromosome 11 may eventually lead the way toward a genetic treatment for the disease," he said. "But in the near future, it may suggest what kinds of drug treatments will work best to increase fetal hemoglobin production in these patients, the ones who are dealing with this problem now."

—Ingrid Heggoy

It is among the most common genetic disorders. You may have it and not even know it.

Thalassemia is a hereditary hemoglobin defect and is closely related to sickle cell anemia. There are over 100 variations of the disease, which range from patients with no symptoms to those who can only survive a few hours after birth.

"Thalassemia is caused by a genetic defect on chromosome 11 or on chromosome 16," said Dr. Titus H.J. Huisman, Regents' professor and chairman of the Department of Cell and Molecular Biology at the Medical College of Georgia.

"Regions of these chromosomes are responsible for making the components of hemoglobin found in the blood. If there is a gene deletion or gene mutation in the areas that make the alpha, beta, gamma or delta molecular chains and insufficient numbers of them are produced, this may cause a decrease in the synthesis of normal hemoglobin and cause thalassemia."

Hemoglobin travels throughout the body in red blood cells, delivering oxygen to organs and tissues. In patients with thalassemia, especially the more severe forms, not enough normal hemoglobin is created and the cells have a shorter lifespan, causing anemia.

When thalassemia causes red blood cells to be destroyed, it releases free iron into the bloodstream, which, left untreated, can cause complications such as cardiac problems, kidney complications and diabetes. The severity of the symptoms depends on how many genes are affected and which variation of the disease a patient has.

"For example, in alpha thalassemia, the body has four genes that make alpha chains," Dr. Huisman said. "If one of them is not there, the body still has three left, and the person can function normally. If two or three are gone, you see symptoms of thalassemia. If all four are gone, it is fatal."

Identification, Please

Different types of thalassemia are caused not only by different genetic defects, but actually by different types of defects, as well.

"Most often alpha thalassemia is caused by gene deletions," he said. "Part of the DNA just isn't there. Beta thalassemia is usually caused by gene mutations."

Beta thalassemia often causes more severe symptoms, according to Dr. Huisman.

MCG is an international reference center for the identification of the different types of thalassemia. Researchers at MCG have discovered about 25 types and work is under way on others.

"We are researching new thalassemia variations, ones we know are different but we don't know what they are yet," Dr. Huisman said. "The variations of this disease are astounding."

"We also have a study now looking at other genetic differences of thalassemia patients. Some people with the same type of thalassemia do better than others with that same mutation, and we're trying to understand why that could happen, and perhaps it could have implications for patient care in the future.

"We have a repository of all the various identified mutations and we have literature about the different types, so physicians, researchers, patients or others can learn about them," Dr. Huisman said.

Physicians and researchers also can send blood samples to the reference center for testing and typing, Dr. Huisman said. The Department of Cell and Molecular Biology serves as a training center for foreign scientists and physicians; at least 40 M.D.s or Ph.D.s from 10 countries have received a year of such training during the past five years.

The center edits a professional journal, *Hemoglobin*, which reports on research related to thalassemia, sickle cell anemia and other abnormalities of globin genes.

—Ingrid Heggoy

Plan Your Giving

The $500 billion plan to reduce the deficit was signed into law recently. The purpose here is not to address the merits of the law, but to provide a brief overview as to how some specific changes in the law may affect your charitable giving.

The change in the tax laws for 1991 which affect the charitable giving patterns of individuals are outlined below. From my readings, most of the experts seemed to think that the law makes such minor changes in tax rates that most donors won't notice any difference. We can also be comforted in knowing that charitable giving is still enhanced by the tax laws. For most people, the tax benefits from donations will be increased, but for others, they may be decreased. As always, each donor will want to check with his personal adviser.

—Bruce Howerton
Director of Planned Giving

	The Change for 1991	***The Effect***
Tax Rates	The top tax rate will be 31 percent. In 1990, the base rate was 28 percent with a 5 percent surtax phased out as income increased. The surtax is eliminated in 1991.	The new rate will reduce the after-tax cost of making a gift from $72 per $100 to $69 per $100. In other words, the tax savings will be increased by $3 per $100 donation.
Limit on Deductions	Taxpayers will reduce their itemized deductions by 3 percent of the amount their adjusted gross income exceeds $100,000. A taxpayer with AGI of $150,000 will reduce deductions by $1,500.	Effects should be minimal. For some whose itemized deductions barely exceed the standard deduction, the reduction could eliminate the need to itemize.
Capital Gains	The maximum tax rate on capital gains for most taxpayers will be 28 percent down from 33 percent.	The lower rate may provide less incentive for some donors, but the ability to give appreciated assets without having to report capital gains is still a tremendous benefit.
Alternative Minimum Tax	Rate is 21 percent in 1990.	Rate will be 24 percent in 1991. Few donors are subject to the alternative minimum tax because of charitable contributions, but for those who are, the tax rate will increase by 3 percent. This could provide a disincentive for making a gift of appreciated assets.

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